

AN INTEGRATED
PRACTICE OF
MEDICINE

AN INTEGRATED PRACTICE OF MEDICINE

*A Complete General Practice of Medicine
From Differential Diagnosis by Presenting
Symptoms to Specific Management of the Patient*

By HAROLD THOMAS HYMAN M D

1184 Illustrations 305 in Color
319 Differential Diagnostic Tables

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Associate Editors WILLIAM PEIFFER M.D.
NATHAN PENSKY M.D.

The senior author wishes to express his gratitude to Dr Nathan Pensky for allowing him to use the Kodachromes from his collection which are reproduced in this section

CHAPTER 111

INTRODUCTION PHYSIOLOGY AND PATHOLOGY METHODS OF DIAGNOSIS

THE tegumentary system includes *skin hair nails sweat and sebaceous glands*. Disturbances of these structures are of universal occurrence and furnish frequent problems for the general practitioner. In point of fact a dermatological paraphrase of Shakespeare's *Seven Ages of Man* might well refer to infantile eczema, acne vulgaris, furuncles and boils, contact dermatoses of occupational and cosmetic origin, venereal diseases, alopecia and senile pruritus.

The majority of skin diseases are being increasingly recognized as local manifestations of systemic disorders. This concept emphasizes the diagnostic implication of the cutaneous phenomenon and directs the attention of the therapist toward a broader program of treatment. *Descriptive dermatology* which concerns itself with the depiction of local damage is often confusing and disappointing so far as therapeutic results are concerned.

The management of skin diseases offers a formidable challenge to the practitioner inasmuch as the patient is an interested and often hypercritical co-observer of progress. Blemishes on visible surfaces, particularly the face, are cosmetic, social, economic and psychologic handicaps. Rarely does the patient arrive for consultation until the popular and widely advertised salves, creams, lotions and ointments have been applied without favorable effects; often the attempted cure has resulted in superimposed inflammatory disturbance of chemical origin.

The treatment of skin diseases is most satisfactory when beneficial results are obvious to patient and practitioner. These happy eventualities may be anticipated if the broader aspects of dermatology inseparably bound up with internal medicine are observed. Failure stalks the therapist who attempts to obtain a result from local and topical applications unaided by measures aimed at underlying systemic conditions. Confusion attends most diagnosis and therapy when a *treatment dermatitis* resulting from idiosyncrasy or overzealousness is superimposed upon a primary skin affliction that was in itself of obscure origin.

DERMATOLOGIC TERMINOLOGY

The approach to clinical dermatology is often so confusing and baffling as to discourage the practitioner from any serious attempt to master its intricacies. This chaos results partly from the terminology which unfortunately substitutes pedanticism for intelligence. Medical management too often consists of telling the patient in Latin or Greek what he has complained of in English. Thus the sufferer reports itching and the diagnosis is *pruritus*; ordinary prickly heat becomes *miliaria rubra* and baldness, *alopecia*.

To complicate matters still further a disease of unknown origin is given a descriptive name such as psoriasis (psora the itch) When a clinically similar but also unknown disease is described it becomes parapsoriasis (like the itch) and another variant becomes prapsoiriasis varioliformis (like the itch resembling smallpox) Again the nomenclature resorts to the use of the name of the dermatologist particularly connected with the disturbance The reader is treated to such diagnoses as Boeck's sarcoid Paget's disease of the nipple Fox Fordyce disease or Bowen's or Kaposi's disease Or the terminology is encumbered with a descriptive adjective indicating the site of the disease such as pubis axillaris vaginalis or one relative to a clinical description such as recurrens for conditions that tend to wax and wane or hiemalis for winter difficulties

The textual material that follows aims to avoid this confusion in terminology by retaining only those older titles which are honored by custom and time

ANATOMY OF THE SKIN

See *Anatomic Review* (p 3498)

PHYSIOLOGY OF THE SKIN

The skin functions as a covering layer to protect the more vulnerable deeper tissues It serves as a vast sensory structure as an excretory and secretory organ and it plays an important role in the metabolism of water and the regulation of body temperature

Protective Functions—The keratin of the horny layer of the skin is a very resistant substance capable of withstanding contact with strong mineral acids In parts exposed to continual pressure as the palms and soles the stratum corneum is particularly thick affording additional protection to relatively superficial tendons muscles nerves and vessels Protection against the harmful effects of the sun's rays is also afforded by the pigment of the skin

Sensory Functions—The sensory functions of the skin relate to the recognition of touch heat, cold and pain These discriminatory faculties of the skin protective in nature serve to warn against harmful influences and are more properly discussed under the abnormalities of innervation (p 3227)

Excretory Functions—The excretory functions are of small importance other than the production of water loss Sodium chloride is present in the sweat as well as minute amounts of alkaline salt, urea and lactic acid Excessive loss of sodium due to profuse sweating may lead to heat cramps and heat exhaustion In severe renal insufficiency with azotemia, the skin vicariously excretes considerable amounts of urea, producing the urea snow Unfortunately this is a feeble and agonal excretory effort Attempts to stimulate the excretory function by the use of hot packs and diaphoretics while well intended, usually serve only to further deplete the patient

Secretory Functions—The secretory function of the skin as a whole is of little significance Sebum produced by the sebaceous glands is not a true secretion but rather a greasy substance liberated by the fatty degeneration of the cells of the glands This material coats the skin and protects its surface Of great clinical significance is the possible relationship between sebaceous glands and the adolescent problem of acne vulgaris The skin glands are modified in the female breast and in the anogenital regions for accessory functions in the processes of reproduction The mammary glands secrete milk while the odor of substances in the erogenous zones are sexually exciting during "heat"

Metabolic Functions—The skin functions importantly as a regulator of body temperature Most of the total heat loss of the body occurs through radiation convection conduction and evaporation of water from lungs and skin With a daily heat loss of 3000 calories approximately 2500 calories are accounted for by the skin controlled by dilatation or constriction of the cutaneous vessels The importance of the skin in the regulation of body temperature is recognized by the practitioner who employs the sponge bath as a most effective and harmless antipyretic measure

Vitamin D is synthesized in the skin by the action of ultraviolet light converts the ergosterol of the skin to vitamin D A little more oxygen absorption and excretion of carbon dioxide

PATHOLOGY OF THE SKIN

The exposed position of the skin permits observation of abnormalities from their inception and through the various phases of evolution and involution. In this respect the skin presents a unique opportunity for the study of the nuances of pathologic change. The disease processes of the skin do not differ essentially from those of other organs of the body. Variations depend upon anatomic differences, particularly in relation to the glandular structures, the hair follicles, the nails and the hair.

Congenital Lesions.—Alterations in the skin may be congenital or acquired. The congenital affections are generally conditioned upon *hypoplasia* (ectodermal defects, albinism) or *hyperplasia* (pigmented nevi, ichthyosis). The congenital diseases of the skin are like those of systemic nature, problems due to some inherited abnormality of the germ plasma. Obviously congenital syphilis cannot properly be placed in this category and the suggested name of "prenatal syphilis" seems more appropriate.

Acquired Lesions.—The acquired affections of the skin may be induced by factors which reach the skin from the outside world as traumas of various sorts and as infections. They may result also from influences acting upon the skin from within the body as a *hematogenous* or *lymphogenous dissemination* of bacteria, allergens, toxins or metabolites.

The Site of Reaction.—Pathologic changes may be confined to the *epidermis* or *dermis*. Most often there is simultaneous involvement of both layers, although the intensity of reaction is usually greatest in the stratum which is primarily affected. At times a disease will affect with exquisite selectivity a single structural element of the skin. Notably this is seen in diseases of pigment formation where the melanoblasts alone may be involved.

Types of Cutaneous Reaction.—The various cutaneous alterations may be dependent upon (1) simple dynamic factors in blood supply as interference with arterial flow or venous return, (2) inflammation which may be acute or chronic induced by mechanical, thermal, electrical or radiant energies or by chemical, bacterial, viral, fungous or parasitic agents, (3) *hemorrhage* which may be minute (petechia) or massive (ecchymosis), (4) *atrophy* or *hypertrophy* of a single element or layer of the skin or of all strata, (5) *anomalies of pigment formation* including an absence, deficiency or excess of elaboration, (6) *neoplastic growths*, benign and malignant and (7) *neuroses* where initially there is only subjective symptomatology without obvious objective alteration.

Special Skin Pathology.—Certain processes in the skin are designated by special pathologic terms.

Hyperkeratosis refers to hypertrophy or thickening of the stratum corneum.

Acanthosis is the term used to describe benign hypertrophy of rete mucosum where cellular outlines and the intercellular bridges are retained and the basal membrane is clean and unbroken. In epithelioma acanthosis also occurs but differs from the benign form. The intercellular bridges are lost, the cell outline is not so distinct, the basal membrane is ruptured and there is downgrowth of the epidermal cell into the corium.

Parakeratosis indicates abnormality of transformation of the cells of the granular layer into horny cells. The swollen horny cells with retained nuclei shed in laminae or scales.

Dyskeratosis refers to a more general state of abnormal cellular development in the epidermis attended by peculiar degenerative changes.

Vesicles and *bullae* are formed by the disruption of epidermal cells after intra- or extracellular edema with the formation of smaller or larger sacs containing serum and blood cells. Bullae may also be produced by separation of the dermis and epidermis through localized accumulations of fluid.

Lichenification is a common secondary change in the skin provoked by prolonged friction, usually scratching. The skin is thickened, its lines are sharply defined, it is grayish or yellowish gray in color, it shows closely crowded polygonal papule-like lesions.

Inflammation is the commonest response to injuries of various sorts. It is a protective and reparative device of tissues. At the site of injury there occurs dilatation of the blood vessels (*erythema*) and an escape of fluid and formed elements from the blood (*edema*). This reaction is purposeful, tending to dilute, destroy and carry off the toxic agent. In more chronic inflammatory processes, especially the granulomas, the changes are not so rapid in pace and the cell types in the exudate are lymphocytes, mast and plasma cells. There is a more marked stromal tissue cell reaction as evidenced by proliferation of connective tissue cells, epithelioid cell formation and the production of giant cells.

The elaboration of protein-digesting enzymes by the polymorphonuclear leukocytes plays

a great part in the liquefaction of dead tissue and bacteria producing pus which is discharged or fragments which are absorbed by the blood and excreted

METHODS OF DIAGNOSIS

The compleat practitioner regards the diagnosis of disorders of the skin as an exercise involving far more than mere observation of the rash and a pontifical enunciation of the main descriptive features in bad Latin or worse Greek. He obtains a history of the origin character distribution and evolution of the eruption inquires into pertinent details of the general health of his patient conducts local systemic and laboratory examinations considers the differential diagnosis of similar eruptions and when necessary consults with the specialist dermatologist

History—The general history often yields valuable information in the recognition of a dermatosis. *Familial predispositions* occur in atopic dermatitis acne vulgaris psoriasis and alopecia. Transmission of infection in syphilis occurs from mother to fetus. Household epidemics are the rule in impetigo dermatophytosis scabies pediculosis and disorders due to dietary deficiencies such as are seen in the avitaminoses. Intertrigo allergic dermatoses, dermatophytoses erythema multiforme and pityriasis rosea are encountered with greater frequency in spring and summer whereas acne vulgaris and psoriasis tend to worsen in the winter.

The influence of age is of such importance that special charts of differential diagnostic features have been prepared for the newborn and infants (p 3146) children and adolescents (p 3360) and the aged (p 3214).

Personal hygiene is often an etiologic factor of considerable importance. Uncleanliness is more frequently seen in those who suffer from scabies pediculosis fungous infections pyoderms and seborrheal dermatitis. In contrast the excessive use of soap and water often produces bath itch and contact dermatitis as the result of hypersensitivity to ingredients of toilettries many patients suffer from seborrhea sicca (dandruff) as the result of too frequent washing of the scalp.

Contact dermatitis (dermatitis venenata) and *drug eruptions (dermatitis medicamentosa)* are probably the skin afflictions most frequently presented to the practitioner. There are best elucidated by a searching history and may be confirmed by patch tests. Greatest suspicion attaches to cosmetics toilettries wearing apparel and substances used in one's occupation. For details the reader is referred to the material dealing with cosmetics (p 3138) and tables of the skin hazards due to occupation (p 4060) and drugs (p 3339).

On a few occasions the practitioner is aided by the history of a previous experience with a dermatosis. *Recurrence* is the rule in psoriasis herpes simplex atopic dermatitis contact and drug eruptions neurodermatitis urticaria dermatophytosis and acne vulgaris.

The *subjective symptoms* that accompany an eruption often provide considerable assistance in differential diagnosis. Painful dermatoses are infrequent but they are of sufficient importance so that their diagnostic features are considered in a separate table (p 3250). The complaint of itching is of great significance because of its high incidence and its importance from the standpoint of symptomatic therapy. Separate tables have been prepared for *generalized pruritus* (p 3170) *localized itching der*

matosa (p 3178) *pruritus ani* (p 1916) and *pruritus vulvae* (p 2594) The treatment of itching is summarized in the *Therapeutic Index* (p 3132 3135 3136)

Few patients consult the physician concerning an eruption until they have tried home remedies or one recommended by a friend or a local pharmacist In consequence the practitioner often is confronted with the complicated problem of an underlying eruption obscured by a superimposed treatment dermatitis and it may be impossible to arrive at any diagnostic conclusion until the effects of it have worn off

Evolution of the Eruption—The history of the course that the eruption has pursued often gives important clues in diagnosis Rashes such as the exanthems and pityriasis rosea disappear wholly and completely within the course of a few weeks whereas angiomas moles and neoplasms may persist unchanged over long periods of time Lesions due to tuberculosis syphilis deep fungous infections precanceroses and malignancies often progress at an almost imperceptible tempo until they assume alarming proportions before the patient seeks professional advice

General Physical Status—In all but the simpler dermatoses a complete physical examination is essential Amongst the systemic disturbances which often manifest themselves by eruptive phenomena are *generalized infections* (p 3245) *avitaminoses* (p 3235) *endocrinopathies* (p 3238) *abnormalities of metabolism* (p 3240) and the syndromes associated with *allergy* (p 3329)

Demonstration of the presence of a systemic disorder does not necessarily establish its relationship to the dermatosis The patient with syphilis or tuberculosis may have *acne vulgaris* or *pityriasis rosea* under which circumstances there is a coexistence of separate and distinct clinical entities The practitioner should gather his facts and then make interpretations according to the merits of the presenting conditions

Quantitative Skin Changes—The skin often presents striking alterations in texture thickness and pigmentation Many of these deviations are within normal limits whereas others are caused by situational circumstances Thus many families possess soft delicate skins which react violently to simple changes such as exposure to sun others are relatively thick skinned as a racial or familial characteristic or as a result of exposure the latter seen most strikingly in farmers and sailors who live their lives in the open Under pathological conditions thickening of the skin is seen in *hypothyroidism* *acromegaly* *leprosy* and *ichthyosis* a hide bound cutaneous surface characterizes *scleroderma* a soft and delicate skin is often noted in *hyperthyroidism* *tuberculosis* and *rheumatic fever* the *senile skin* is thinned parchment like and wrinkled with scattered pigmentations and keratoses In each of these circumstances the dermatologic manifestation is overshadowed by the more fundamental cause

Eruptions—Eruptions represent qualitative changes in the cutaneous surfaces They may be localized or generalized the latter have led clinicians to speak of the skin as the mirror of body

The *primary* lesions of the skin include macules papules vesicles pustules bullae nodules tumors and wheals The *secondary* lesions are scales crusts excoriations fissures rhagades ulcers scars and stains Additionally the skin may present comedones miliums burrows punctums eryth

a great part in the liquefaction of dead tissue and bacteria producing *pus* which is discharged or fragments which are absorbed by the blood and excreted

METHODS OF DIAGNOSIS

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suggest the specific character of the disturbance. The chancre of syphilis and the vesicle of smallpox are notably *shotty* and *indurated*; purulent lesions fluctuate and gummas have an *elastic* feel. The skin in scleroderma is *thickened* and *inelastic*; the tissue in a chronic cellulitis is *firm* and *brawny*; edematous tissues pit on pressure.

At times the nature of the contents of the lesion may throw light on the nature of the disturbance. The vesicle of herpes zoster or chickenpox contains *straw-colored fluid*; the content of the lesion in smallpox is *purulent* from the sebaceous cyst; a *caseous* substance is expressed from purulent lesions; yield material that may be *thin* and *opaque* or *thick* and *creamy*; the discharge from the lesion of glanders has a characteristic *chocolate* like appearance.

Incidence of the Dermatoses.—The practitioner discouraged by the multiplicity of described dermatologic conditions may take solace in the fact that more than 90 per cent of consultations deal with a *relatively few skin conditions*. These include in alphabetical order the following: acne vulgaris, alopecia (baldness), atopic dermatitis (infantile eczema), callositas (callus), chilblain, clavus (corns), contact dermatitis (dermatitis venenata), drug eruptions (dermatitis medicamentosa), hirsuties (hairiness), keratoses, lichen planus, moles (nevi), intertrigo, herpes simplex, herpes zoster, pruritus (itching), psoriasis, pediculosis, pyoderma (furuncles, carbuncles and impetigo contagiosa), ringworm (dermatophytosis), scabies, seborrhea, seborrheal dermatitis, sunburn (solar dermatitis), syphilis, neurodermatitis, verrucae (warts) and urticaria (hives).

Dermatologic Atlas.—Since the diagnosis of skin disorders is primarily a visual exercise, the present Section is profusely illustrated. Most of the colored photographs have been taken from the collection of the Associate Editor Dr. Nathan Pensky, to whom we are greatly indebted.

The illustrations of the present Section constitute a dermatologic atlas. They have been distributed throughout the text so that each picture might appear near to the material to which it refers. For convenience the following alphabetical list has been prepared for purposes of reference.

Atlas of Dermatology

Indicates colored plates

- | | |
|---|---|
| Anthrax axillary sweat gland Fig 949H | Anemia macrocytic hyperchromic Illustrian |
| p 3247 | glossitis in Fig 347 p 1674 |
| pericoronal on partly erupted third molar | Angina scarlatinal strawberry tongue in |
| Fig 366 p 1696 | Fig 20 p 17 |
| Acanthosis nigricans Fig 985 p 3356 | Angioma cavernous Fig 914 p 3200 |
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| rosacea Fig 986A p 3359 | Arsenic dermatitis Fig 107 p 193 Fig 9 |
| vulgaris Fig 986C p 3359 | p 351 |
| Acrodynia Fig 886 p 3143 | Atopic neurodermatitis Fig 982 p 3331 |
| Actinomycosis Figs 965 p 3310 75 p 486 | Atopy due to drug Figs 982 983 p 3331 |
| Addison's disease pigmentation in Fig 346 | Atrophy macular due to syphilis Fig 993 |
| p 1674 | p 3103 |
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| en up de sabre Fig 1007B p 344 | Beriberi et Fig 937 p 306 |
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| Ammonia lichen Fig 971 p 3317 | Blastomycosis Figs 75B p 486 906 p 3311 |

emiss petechiae ecchymoses purpuric spots telangiectases erythrodermia gangrene and pigmentation

These lesions are defined as follows

- Macule** A circumscribed discoloration of the skin that is neither elevated nor depressed
Generalized (p 412) *Localized* (p 3390)
- Papule** A solid elevated formation of the skin no larger than a split pea
Generalized (p 412) *Localized* (p 3390)
- Vesicle** An elevated formation of the skin containing free serous fluid and no larger than a split pea
Generalized (p 422) *Localized* (p 3334)
- Bulla** An elevation of the skin larger than a split pea and containing free fluid (p 3334)
- Nodule** A solid formation of the skin larger than a split pea and smaller than a hazel nut (p 3210)
- Tumor** A solid formation of the skin larger than a hazel nut (p 3210)
- Wheal** A transitory circumscribed elevation of the skin produced as the result of edema of the corium often with pseudopodia demonstrable at its borders (p 3316)
- Scale** A mass of exfoliating or exfoliated epidermis (p 3382)
- Crust** A mass formed on the surface of the skin from the accumulation of dried exudate or debris
- Excoration** Superficial abrasion of the skin (p 3186)
- Fissure or Rhagade** A crack in the skin usually extending through the epidermis into the corium (p 3218)
- Ulcer** A circumscribed loss of substance of the skin extending through epidermis into corium (p 3218)
- Scar** A connective tissue replacement of loss of substance of the corium
- Comedone** An inspissated plug of sebum and detritus filling a hair follicle (*blackhead*) (p 3358)
- Milium** A ball like pinhead collection of sebum seen chiefly on the face (*whitehead*)
- Burrow** A tract or gallery made as a passage way in the skin by certain parasites notably the *Acarus* of scabies (p 3181)
- Punctum** A tiny central hemorrhagic point usually the result of an insect bite (p 3180)
- Erythema** Dilatation of the blood vessels producing redness and heat in the skin In active erythema there is dilatation of the vessels and increased blood flow In passive erythema there is stasis in the vessels with dusky red cyanosis of the skin and coldness
Generalized (p 422) *Localized* (p 3162)
- Petechiae** Pin sized hemorrhages into the skin or mucous membranes In subacute bacterial endocarditis the petechiae are characteristically white centered (p 3398)
- Purpura** Skin hemorrhages of larger size than petechiae (p 3422)
- Ecchymosis** A bruise that is larger than the purpuric spot and usually deeper extending into the subcutaneous tissues
- Telangiectases** Visibly and permanently dilated superficial capillaries (p 3391)
- Erythrodermia** A generalized or universal redness of the skin Usually accompanied by scaling and vesicle formation
- Gangrene** Actual necrosis of the skin with atrophy ulceration or actual amputation
- Pigmentation** Discoloration of the skin due to the deposition of abnormal coloring matter
Generalized (p 3212) *Localized* (p 3154)

Distribution of the Lesion—The distribution of the commoner lesions serves as a diagnostic aid Many of the dermatoses have a predilection for appearing in certain areas of the body At times the elective localization is understandable such as the presence of pyogenic lesions and contact dermatitis on the exposed parts or of fungous infections in the folds of the skin where friction furnishes moisture and epithelial detritus suitable for growth of the micro organism In other instances localization of the dermatosis has no rational explanation

As an aid to *differential diagnosis* tables have been prepared according to the areas of predilection in which the various dermatoses appear The complete roster of tables is listed on p 3110

Consistency and Content of Lesion—The consistency of the lesion may

- Glomus tumor Fig 920 p 3 01
 Glossitis Hunterian in macrocytic hyperchromic anemia, Fig 317 p 1674
 in niacin deficiency Fig 940 p 3237
 Grain itch Fig 909 p 3194
 Granuloma annulare* Fig 991 p 3335
 furunculosis Figs 90 993 p 333
 inguinal Donovan bodies in Figs 73 p 47
 lesions of penis groin and anus Fig 959 p 3 77
 of vagina Fig 617 p 2391
 Gum lymphosarcoma of Fig 381 p 1718
 HALO SATURNINUS Fig 332 p 1675
 Hemangioma, Fig 913 p 3200
 Hemolymphangioma Fig 912 p 3200
 Hemorrhages capillary in adult scurvy due to vitamin C deficiency Fig 914 p 3 39
 Herpes labialis, Fig 344 p 1671
 simplex of lip* Fig 961C p 3 89
 of penis Fig 961F p 3 89
 zoster in gangrenous stage Fig 961B p 3289
 in vesicular stage Fig 961A p 3259
 Herpetiform lesions, Figs 94 p 3241 961 p 3 89
 Histoplasmosis Fig 96F p 437
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 Hyperkeratosis follicular of vitamin A deficiency Fig 934 p 3236
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 KALA AZAR liver with Leishmania donovani in Fig 89 p 323
 Kaposi disease Figs 9 3 9 9 p 3221
 keloid Fig 892 p 3160
 keratosis blenorrhagica Fig 9 9 p 3258
 hyperkeratosis of vitamin A deficiency Fig 934 p 32 6
 kerle Fig 902 p 3 01
 Koplik spots of measles Fig 64 p 410
 Leukemia lymphatic black tongue in Fig 367 p 1696
 Leukoplakia Figs 921 p 3201 362 p 1691
 Lichen planus Fig 994 p 3397 Fig 338 p 1669
 simplex Fig 806 p 3190
 Lip epithelioma of Fig 380 p 1718
 Fordyce disease Fig 1010 p 3465
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 Lupus erythematosus Fig 995 p 3397
 acute disseminated Fig 997 p 3400
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 Lymphorathia venereum* Fig 937 p 3277
 Lymphosarcoma of gum Fig 381 p 1718
 MADUROMYCOSIS, Fig 909 p 3314
 Magenta tongue of vitamin B deficiency with glossitis Fig 343 p 1674
 Measles Koplik spots Fig 64 p 410
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 Melanocarcinoma with distant metastases Fig 9 9 p 3221
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 Meningitis meningococcal purpuric rash of Fig 23 p 212
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 Molluscum contagiosum generalized Fig 961D p 3289
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 Mondras Fig 96E p 437 1009B p 3453
 in a diabetic Fig 962E p 3294
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 Mucous patch on tongue Fig 343 p 1671
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- Bowen's disease * Fig 925 p 3221
 Brucellergen skin reaction * Fig 45 p 318
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 Bullae in erythema multiforme Fig 989B p 3375
- CAPILLARY NEVUS (strawberry mark) Fig 911 p 3900
 Carbuncle before and after penicillin * Fig 949 p 3247
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- Endocarditis subacute bacterial Fig 921 p 1092
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 Erythema induratum * Fig 953E p 3963
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 Excoriations neurotic Fig 939 p 3939
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- FACE erysipelas of Fig 19 p 165
 Favus * Fig 962G p 3994
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- Scrub typhus primary lesion of Fig 59 p 382
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- capillary hemorrhages due to * Fig 944 p 3239
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Laboratory Aids in Diagnosis—The diagnosis of many dermatoses may be confirmed or established by laboratory aids. The greatest assistance is obtained by histologic, bacteriologic, serologic and immunologic tests and by studies of the blood.

Biopsy—Biopsy of a skin lesion is easily obtained but dermatologic histopathology is less readily mastered. The practitioner should refer biopsy studies to the specialist, particularly in chronic dermatoses that may possibly be of an infectious or neoplastic nature.

Bacteriology, Serology and Immunology—The diagnosis of a skin disease may be suggested or proved by the studies of direct smears, by bacteriologic cultures, by serologic and skin tests or by animal inoculation.

Hemograms—On occasions, particularly in infectious mononucleosis and leukemia, the nature of the eruption is revealed by the hemogram or marrow smear. Confirmation of findings necessitates review of the slide by the clinical pathologist.

Psychic Overtones—Most dermatoses involving visible surfaces become associated with psychic overtones of greater or lesser intensity. In the adolescent acne vulgaris produces a marked sense of inferiority and may lead to a pathologic degree of introversion. Birthmarks, hypertrichosis and the like may bring about character changes of considerable importance in the sensitive. When these progress to an extreme degree, it may be necessary to undertake unusually active local therapy and consider consultation with the psychiatrist.

Severe and protracted itching has a particularly depressing effect when it is localized to anus and genital areas. Under these circumstances, psychiatric influences undoubtedly play a major part in initiating as well as

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used for urethral irrigation in gonorrhea. Wet dressings (1:1000) or an ointment of 1 per cent proflavine oilate in equal parts of petrolatum and calcium carbonate are recommended in *furunculosis secundarily infected eczemas* or *monilial infections*.

Neutral Acriflavine in 0.1 per cent strength is a component of triple-dye used in burn therapy.

Adrenalin—See Epinephrine.

Alcohol U.S.P.—Alcohol is a *fat solvent*, a *rubefacient*, an *astringent*, a *refrigerant* and an *antiseptic*. It is employed to prepare the skin prior to surgical procedure.

As a *rubefacient* and *astringent*, alcohol is the main ingredient of most "rub-lin" lotions, after-shave preparations and toilet waters. The concentration under these circumstances varies between 40 and 70 per cent.

Alcohol is used as a *menstruum* for other active drugs, as for example resorcinol in *scalp lotions*.

Aloe Vera—The leaf of the plant, *Aloe vera*, has been used with success in the treatment of ulcers following roentgen ray and radium dermatitis. The leaf consists of two distinct laminae between which is a jelly like substance. The jelly is applied to the ulcer on gauze and is bandaged in place.

The Russians suggest that cut aloe leaves be laid on a plate covered with gauze and placed in a darkened icebox for fifteen days. At the end of this time the leaves are cleansed with an alcoholic solution of brilliant green and small thorns are cut off. One hundred grams of the leaves are finely cut with a scissors and ground in a mortar with a pestle. Three hundred cc. of distilled water are added. The mixture is allowed to stand for an hour after which it is filtered through paper, poured into ampoules and sterilized daily for three days at 60°C for one hour. Between and after sterilizations ampoules are kept in the icebox.

Tissue therapy with aloe leaves appears to be successful in the treatment of *keratitis lupus vulgaris*, *tuberculous ulcers of the skin*, *lupus erythematosus*, *scleroderma*, *psoriasis* and *acute ulceration cutaneous leishmania*.

Alum U.S.P.—Aluminum aluminum salts are *astringent* and *otic type* and *mildly antiseptic*. Dried or burn alum is an astringent dusting powder. Alum in 1 to 5 per cent solution is used as a gargle. Wet dressings of aluminum acetate (*Buro's solution*) are mildly astringent and antiseptic when diluted with water 1:10 to 1:20.

Solution of aluminum subacetate N.F. is 8 per cent strength. Diluted 1:10 to 1:20 with water it is a *mildly astringent and otic and antiseptic wet dressing*.

As an *acidotic* the following is recommended:

| | |
|---------------------|---------|
| R Aluminum chloride | 120-300 |
| Glycerin | 36-60 |
| Water | |
| Alcohol as qs ad | 1200 |
| Dab on locally | |

Aluminum—See Alum.

Ammoniated Mercury—See Mercurials.

Androgen—Androgen preparations are available in oil or t form. They have been used successfully in the treatment of *senile pruritus* and *pruritus vulvae*.

Anesthetic—See Benzocaine.

Antialnetic Ointment—See Chrysarobin.

Argyrol—See SI.

Asol—See Phenol Grop.

Asphen—Arphenamide U.S.P. (salicylanilic acid) is a *phenol derivative* in 1 to 10 per cent solution in glycerin in the local treatment of *apical infections*. They are especially recommended in *lymphatic infections* (*tusosporilosis*) of the throat, gums and genitals. Local use on the syphilitic chancre is to be deprecated since syphilis is a generalized disease to be treated systemically (p. 340).

Az chloram d.—See Chlor.

THE TEGUMENTARY SYSTEM PHARMACOTHERAPY OF SKIN DISEASES COSMETICS

THE treatment of diseases of the skin constitutes a specialized field. In consequence the material concerned with the local and topical application of drugs and biologicals has been separated from the main section on Pharmacology (p. 3799) and comprises the present chapter.

THE PHARMACOTHERAPY OF THE DRUGS AND PREPARATIONS AVAILABLE FOR LOCAL OR TOPICAL APPLICATION TO THE SKIN AND ACCESSIBLE MUCOUS SURFACES

An imposing roster of drugs and chemicals is available for local or topical application to the tegumentary system and the accessible mucous surfaces. While a few of the more commonly used prescriptions are appended with the list of pure drugs, the practitioner should employ the *simplest and least complicated preparation*. No small part of the difficulty of managing the disturbances of the tegumentary system arises from the fact that complicated prescriptions with many active ingredients are used. Some of these in excessively strong concentration cause a *treatment dermatitis*. Others are employed in a dilution so weak that the effective result is not obtained.

Particularly in dermatology the reaction to treatment should be regarded as a *variable*. The response to any therapeutic agent is tested in a probatory way. The strength of the ingredient may always be increased if a satisfactory result is not obtained, whereas the use of an excessively irritating concentration complicates the therapeutic regimen by superimposing upon the underlying and original lesion a chemical inflammation.

The employment of the potentially irritating preparations should be postponed *until the less irritating agencies have been tried*. When and if it becomes necessary to institute more vigorous therapy, the first consideration is the avoidance of local tissue injury.

Certain therapeutic procedures capable of inflicting temporary or permanent damage are within the province of the *specialist*. These modalities include the application of *caustics*, *roentgen rays* and *radium*.

Acetyl beta methylcholine Chloride NNR—An aqueous solution of the chloride of acetyl beta methylcholine produces vasodilatation when applied by iontophoresis (p. 3792). A 0.2 to 0.5 per cent solution of a proprietary Mecholyl Chloride is placed on the positive electrode. A current of 5 to 30 ma. is used for 5 to 30 minutes. Varying success has been reported in the treatment of *vasospastic disorders*, *scleroderma* and *indolent ulcers*.

A proprietary ointment Imadyl is also available as a *rubefacient*.

Acriflavine—Acriflavine, a yellow dye derived from coal tar, has *antiseptic* and *bacteriostatic* properties. It is comparatively nontoxic and nonirritating. Fresh solutions are prepared and those over one week old should be discarded.

Suppurating wounds are treated with wet dressings or irrigations of 1:1000 solutions in physiological saline solution. Weaker solutions (1:4000 to 1:10,000) are

- | | | |
|---|----------------------------------|----------------|
| N | Soft Soap | |
| | Ethyl Alcohol | |
| | Benzyl Benzoate | aa q.s ad 20.0 |
| | Sg Local use in scabies (p 3181) | |

Betanaphthol—Betanaphthol applied externally as an ointment of 1 to 10 per cent strength is a powerful antiseptic, *leucolytic*, parasiticide and antipruritic. It may be exceedingly irritating to the skin and if absorbed it may produce marked renal irritation.

For strong keratolytic effect
Use as Peeling Ointment

- | | |
|-----------------------|---------------|
| 1, Betanaphthol | |
| 1 precipitated Sulfur | |
| Green Soap | |
| Coll Cream | aa q.s ad 100 |

In the treatment of scabies (p. 3181)

- | | |
|-----------------|------------|
| R Betanaphthol | 4.0 |
| Balsam of Peru | 8.0 |
| Sulfur Ointment | us ad 1200 |

Bismuth Subgallate and Bismuth Subnitrate USP—Bismuth Subgallate (Dermatol) and Bismuth Subnitrate are employed on the skin as *dusting powders* or *ointments* (10 per cent). They are mildly *astringent* and *antiseptic* but are used chiefly for their *styptic* power on open wounds. They form a crust beneath which the wound heals.

Bismuth Tribromophenate (Xeroform)—Bismuth Tribromophenate (Xeroform) is a mild soothing *antiseptic* used in 5 to 10 per cent strength in ointments or lotions. It may be used full strength as a dusting powder and may be combined with 5 to 5 per cent ammoniated mercury ointment, in the treatment of *impetigo contagiosa* and *impetiginized eczemas*.

Bo ax—See Sodium Borate

Boric Acid U.S.P.—Boric Acid is a mild antiseptic and astringent. In a saturated 5 per cent aqueous solution it is employed as a wash or a lotion particularly for the wet dressing the collyrium and for irrigation of the bladder. Boric acid powder is employed as dusting powder alone or combined with starch or talcum.

The official 10 per cent Boric Acid Ointment is mildly antiseptic. It is used chiefly as a protective dressing.

B Liant Green—B Liant Green is a dye used locally in pustular affections of the skin. It is a component of the triple-dye preparation (p. 3981) recommended for the treatment of burns. It may be applied in 1 to 2 per cent aqueous solution on moist eczemas, furuncles and pyogenic infections of the skin. It can also be incorporated into ointments and pastes.

B was Solutio. —See Al m

B tes. —See Bel cane

Calamine (Prepared)—1 prepared calamine is powdered zinc oxide containing a small amount of ferric oxide. Calamine is popularly used in a *lotion* for protective and drying purposes and in the official calamine liniment NF which is oily and protective.

Calamine Liment

- | | | |
|---|-------------------|------------|
| R | Prepared Calamine | 80 |
| | Zinc Oxide | 80 |
| | Olive Oil | 500 |
| | Lame Water | qs ad 1000 |

Balsam of Peru USP—Balsam of Peru is an *epithelial stimulant* and *parasiticide* particularly useful in the treatment of *scabies*

As an epithelial stimulant the balsam is employed in the prevention and treatment of *indolent sores and ulcers*. For this purpose it is best mixed with equal parts of castor oil and applied directly to the pressure spot

A popular ointment used in the treatment of *scabies* consists of

| | |
|---------------------|------------|
| ℞ Balsam of Peru | |
| Precipitated Sulfur | |
| Precipitated Chalk | |
| Soft Soap | aa 48 |
| Petrolatum | qs ad 1200 |

In obstinate cases the balsam content may be doubled

Belladonna—Belladonna is employed locally as the Belladonna Plaster USP or the official Belladonna Ointment USP containing 10 per cent of dried extract of belladonna. It is described as a *local anodyne*. It is extremely unlikely that any specific analgesic effects result from the use of the alkaloid in the plaster or ointment. There is also the possibility that the sensitive individual may develop belladonna poisoning from absorption (p 3875). No useful purpose is served by continuing the official uses of belladonna externally. See *Atropine* (p 3876).

A belladonna like relaxation of the bronchial muscles in asthma sometimes is obtained by smoking *asthma cigarettes* made of stramonium

Benzine and Ether—Benzine and ether with alcohol are used as *cleansing agents* for the skin. Benzine and ether remove gross particles and dissolve fatty material allowing the more effective use of antiseptics. Alcohol (in 70 per cent solution) destroys ordinary surface bacteria.

Benzine and ether are particularly useful in removing adhesive tape. Benzine is made *noninflammable* by mixture with 60 per cent carbon tetrachloride.

Benzocaine (Ethyl Aminobenzoate) USP—Benzocaine or Anesthemin USP is a valuable local *anesthetic*, *analgesic* and *antipruritic*. It is employed in 3 to 10 per cent concentration in dusting powder, ointment, suppository or a lotion.

A useful *paste* in the treatment of *painful ulcers* is undernoted

| | |
|---------------------|------|
| Benzoic Acid | 20 |
| Ethyl Aminobenzoate | 100 |
| Chloroform | 200 |
| Glycerin | 1000 |
| Tragacanth powder | 1000 |

Closely allied chemicals are available in commercial preparations widely used for their local anesthetic action in painful *sunburn* and in *pruritus*. Of chief importance are *butein*, *buteinn picrate*, *nupercaine* and *orthoform*. These may produce widespread dermatitis in *idiosyncrasy*.

Benzoic Acid USP—Benzoic acid is a mild *fungicide*. It is used chiefly as a component of the official Compound Ointment of Benzoic Acid NF, commonly known as "Whitfield's Ointment". The latter contains 12 per cent benzoic acid and 6 per cent salicylic acid in a base of wool fat and white petrolatum.

Benzoin USP—Compound Tincture of Benzoin of approximately 10 per cent strength is a soothing *inhalant* and a mild *expectorant* when added to the steam kettle in tea spoonful doses. Employed externally it serves as a *protective* and *epithelial stimulant*. For the latter use it is painted on *ulcers*, *sores*, *fissures* or *cracked nipples*. As a protective it is applied to the skin prior to the use of light adhesive strapping. Under these circumstances the benzoin often prevents chafing and excoriation.

Benzoated Lard USP—Benzoinated Lard is an *ointment base* which does not become rancid. It is of particular value in scalp preparations.

Benzyl Benzoate—Benzyl Benzoate has found use as a *parasiticide*. Local application is free from messiness and one or two treatments are often successful.

Ca on Oi (Lime Lament)—Carron Oil is an emulsion of linseed oil and lime water. Its use as a *protective* on burns is not recommended because of frequent secondary pyogenic infection beneath the oily film.

C stellar s Paint—See Resorc ol.

Castor Oi USP—Castor Oil is used externally as (1) a *protective* as in the conjunctival sac (2) in the process of *softening the skin* over areas where pressure sores may occur (3) in *scalp lotions* to overcome dryness and scaliness.

Castor oil and balsam of Peru in equal parts make a soothing and stimulating mixture useful in the treatment of *bed sores*.

Chloral Hydrate USP—Chloral Hydrate mixed with an equal part of camphor is employed locally as a *rubefacient* in many popular "rubdown" mixtures and as an *ant pruritic*. It is questionable whether the external use of chloral hydrate is of significant value.

Chlorine—Chlorine gas is a powerful *antiseptic* used to kill micro-organisms in air and water. It is too irritant for use in the treatment of patients.

Dakin's solution consisting of diluted sodium hypochlorite solution and a buffer substance is very valuable in *cleaning necrotic wounds* infected with anaerobic organisms.

Chloramine T and Dichloramine-T are less irritating substitutes for Dakin's solution. The former is used in 0.1 to 4 per cent aqueous solution or as a 1 per cent paste in a base of 15 per cent sodium stearate and 85 per cent water. Chloramine-T is recommended to remove irritant and vesicant war gases (mustard gas and lewisite) from the skin. It is usually prepared as an ointment in a base of vanishing cream. Dichloramine T is used in freshly prepared solutions of 2 to 10 per cent in chlorinated paraffin (*chlorosane*) as a *wound dressing*.

Azochloramid contains about 98 per cent of N-N-dichloroazodicarbonamide and is used in a manner similar to chloramine-T and dichloramine-T. Solutions of 1:1600 and 1:3300 in isotonic buffered saline are used for *dressings*, *packings* and the *irrigation of infected wounds and cavities*.

Chloroform USP—Chloroform used externally as the popular chloroform liniment is an effective *rubefacient* and *counterirritant*. It has been recommended for local application to *insect bites* and *stings* to relieve intense itching.

Chromic Tincture USP—Crystals of Chromium Trioxide are used for *local hemostasis* particularly in *epitheliomas* from septal ulcers. It is also employed for *destruction of local neoplasms*. Its use for the latter purpose is not recommended since control of destruction is uncertain and difficult. It is used by the dentist in the treatment of *Vincent's infection* of the gums.

Cinnabar—See Mercury.

Cocaine—Cocaine and its salts and their substitutes are fully described in the chapter on *Minor Surgery* (p. 3913).

Cod Liver Oil USP—Cod Liver Oil has been used externally for the treatment of burns, but there are better agents for this purpose. In the form of an ointment it is employed in the therapy of *indolent ulcers*, *radiodermatitis* and *tuberculous lesions of the skin*. The usual preparation is 10 per cent of the cod liver oil in ointment of zinc oxide, but the cod liver oil may be used in much greater concentration. A well known proprietary preparation (*Dinitron ointment*) consists essentially of cod liver oil.

Croton Oil—See Oil of Turpentine.

Cold Cream Rosin Water Ointment USP—Cold Cream used as an *emollient* and ointment base in which active ingredients are incorporated. The USP preparation contains no perfume and may be used by patients sensitive to scents.

Chrysarobin USP—Chrysarobin is a *keratolytic* and *keratoplastic* and is used almost as a specific for *psoriasis* and is valuable in the treatment of *chronic inflammatory indolent skin diseases*. More active than tar chrysarobin

| | | |
|---|-----------------|------------|
| I | Therol | 02 |
| | Oil of Bergamot | 0.5 |
| | Tragacanth | 10 |
| | Zinc Oxide | 60 |
| | Calamine | 60 |
| | Olive Oil | 330 |
| | Distilled Water | qs ad 1000 |

Calamine Lotion

| | | |
|---|-------------------|------------|
| ℞ | Prepared Calamine | 80 |
| | Zinc Oxide | 80 |
| | Glycerin | 20 |
| | Lime Water | qs ad 1000 |

The lotion or liniment may be modified for many purposes particularly as an *antipruritic* (p 313C) by the addition of 0.5 to 1 per cent menthol and/or 0.5 to 1.0 per cent phenol

Camphorate d Calamine Powder

| | | |
|---|--------------------------|-----|
| ℞ | Camphor | 0.5 |
| | Menthol | 0.5 |
| | Alcohol q.s. to dissolve | |
| | Calamine | 600 |
| | Zinc Oxide | 600 |
| | Powdered Corn Starch | 600 |

Calamine Ointment N F

Calamine Ointment N F is composed of 17 per cent of prepared calamine in a base of yellow wax wool fat and petrolatum

Calcium—Calcium Hydroxide USP (slaked lime) is used externally at the official Lime Water (U.S.I.) This solution contains approximately 0.15 per cent calcium hydroxide. It is employed as a mild *anatacid* and *astringent* but is more commonly used with oils in the preparation of *emulsions* (see calamine liniment) and in the preparation of *lotions*.

Solution of Sulfurated Lime N F (*liquor calcis sulfuratæ 1ℓ mancezæ solution*) is of value in the treatment of *acne vulgaris* and *extensive pyoderms*. It is prepared by boiling together lime sulfur and water. In *pyoderma* or *pustular folliculitis* it is used in the bath or as a *wet dressing* diluted 1:10 to 1:20 with water.

Calcium Chloride is used to remove *irritant war gases* (mustard gas and lewisite) from the skin. It is prepared as a *paste* mixed with water or it may be incorporated in white petrolatum as an *ointment*.

Calomel—See *Mercurials*.

Cantharides—Cantharides or "Spanish Flies" is an intense *skin irritant*. It produces local congestion and blistering effects which can be carried out much more safely by other modalities. The official preparation is the *Blistering Stearate NF* which contains 3 per cent of cantharides. It is also prepared as a *plaster*.

Cantharides is the main ingredient of most proprietary *aphrodisiacs* for local use; its employment is not without danger because of possible renal irritation. It is also the principal substance in many proprietary *hair tonics*. While it is extremely doubtful whether it is of any value for the prevention or cure of baldness, it may be prescribed as follows:

| | | |
|---|----------------------------|------------|
| ℞ | Tincture of Cantharides | 120 |
| | Castor Oil | 60 |
| | Alcohol | qs ad 1200 |
| | Sig: Local use in alopecia | |

Carbon Dioxide USP—Carbon Dioxide snow may be purchased as "dry ice." Powdered and made into a slush with acetone, it is powerfully *keratolytic*. This slush is employed by the expert in the treatment of *acne pitting*. The solid "snow" is used to destroy local lesions of *acne vulgaris* and *keratos*.

D mazon—See Scarlet Red

Dreuw O niment—See Chrysarobin

Epinephrine (Adrenal) U.S.P.—The important local action of epinephrine is *constriction of small blood vessels*. It is used with *local anesthetics* to delay their absorption and thus prolong action. It also diminishes hyperemia of the conjunctivae, reduces swelling of the nasal mucous membrane and arrests local mucous membrane hemorrhage. It has been successfully applied as a spray to the larynx in 1:100 dilution to alleviate *asthmatic paroxysms*.

Epinephrine produces *mydriasis* and blanching of the membrane when instilled into the conjunctival sac. The pupillary dilatation is an adrenergic effect and is not accompanied by paralysis of accommodation such as occurs with atropine.

Epinephrine substitutes are similar in action but have fewer systemic effects. They include ephedrine sulfate 2 per cent, neosynephrine 0.5 to 1 per cent, propadrine 1 to 2 per cent, amphetamine sulfate (Benzedrine) 1 to 2 per cent and privity 0.1 per cent.

Epsom Salts—See Magnesium Sulfate

Estrone N.N.R.—Estrone, a crystalline estrogenic steroid derived from pregnant urine, may be used locally in the treatment of pruritus vulvae, senile vaginitis and leukorrheal vaginitis in children.

For *pruritus vulvae*, an ointment containing 1000 IU (0.1 gm) per cc is generally prescribed. For *senile vaginitis*, suppositories containing 5000 IU (0.2 gm) are given. For *gonorrheal vulvovaginitis* in children, suppositories containing 200 to 2000 IU (0.05 to 0.2 gm) are inserted daily.

Ethyl U.S.P.—Ether is employed locally as a *cleansing agent* in the preparation of the skin for surgical procedure. The usual technique of the operating room calls for scrubbing of the skin with benzene alcohol and finally ether before the application of the antiseptic solution.

Ethyl Amobenzoate—See Benzocaine

Eugenol—See Oil of Cloves

Fliht—See Pyrethrum

Foam Powder—See Talc

Formaldehyde G.P.—Formaldehyde is a gas. In medicine it is used mostly in the form of an aqueous solution (37 per cent) to *disinfect* metals and fabrics, *preserve* pathological specimens and *fumigate* rooms. Its vapor is very irritant to the mucous membranes of the respiratory tract. Its repeated contact with the skin is frequently followed by redness and hardening. It may give rise to a persistent *eczema* in pathological states.

A substance which depends for its action on the liberation of formaldehyde in acid solution is methenamine. Methenamine tablets burn with a slow flame and may be used to boil water in a spoon or test tube.

Gentian Violet—Gentian violet is an aniline dye (Methylrosaniline) prescribed in 1 to 3 per cent aqueous or alcoholic solution in the treatment of *fungus infections* of the skin and mucous membranes. It is particularly effective in disturbances caused by yeasts as *Monilia albicans*. It is also given orally as methylrosaniline chloride in enteric-coated tablets containing 0.032 gm ($\frac{1}{4}$ grain) in the treatment of *pinworm*.

Gentian violet in 2 per cent strength is part of the triple-dye preparation used in *burn therapy*.

Glycerin U.S.P.—Glycerine is a *vehicle*, a *solvent*, an *emollient* and a mild *astringent*. In the latter capacity it is used in *lozenges* and *rectal suppositories*. As a solvent it is employed in preparing *glycerites*. It is also used in ointment preparations as a *sweetening* and *thickening* agent.

Glycerin Suppository U.S.P. contains approximately 5 gm of glycerine.

Hydrogen Peroxide U.S.P.—The official solution of hydrogen dioxide is of approximately 3 per cent strength. Hydrogen peroxide was formerly used to a great

must be used with *great caution* particularly in the region of the eyes where it may cause violent conjunctivitis. Its use over very wide areas is not recommended since too great absorption may induce *renal irritation*.

Chrysarobin stains the skin brown the hair a greenish yellow the nails a reddish brown clothing a walnut brown. The stain can be removed with a dilute solution of caustic soda.

It is advisable to start therapy with concentrations as dilute as 0.1 per cent and gradually increase the strength. Very dilute concentrations of this drug are often surprisingly effective. The official USP ointment is 6 per cent. Other preparations include the following:

| | |
|---|-----------|
| R (Dreuw) | |
| Salicylic Acid | 0.6-3.0 |
| Chrysarobin | 0.6-3.0 |
| Tar | 1.5-3.0 |
| Soft Soap | |
| Anhydrous Lanoline | aa 30.0 |
| Sig. X use | |
| R Chrysarobin | |
| Traumaticin | 1.5 |
| Sig. Apply to local areas with camel's hair brush | q.s. 30.0 |
| Dispense in rubber-stoppered bottle | |

Anthralin (dihydroxyanthranol) is a proprietary preparation very similar to chrysarobin. It is much more active than the latter hence treatment is usually begun with strengths of 0.1 per cent.

Collodion USP—Collodion is used as a *protective and vehicle*. The Flexible Collodion USP contains 2 per cent camphor and 3 per cent castor oil. Corn collodion which contains 10 to 20 per cent salicylic acid is a valuable *keratolytic agent*.

Copper Sulfate USP—Copper Sulfate is used as a *soft caustic* in the treatment of trachoma. In collyria concentrations of 1:1000 to 1:100 are *astringent and mildly antiseptic*. Solutions of copper sulfate are also used in the treatment of *indolent ulcers* by iontophoresis.

Cuprex a proprietary solution of a copper compound in organic solvents destroys lice and their nits. It can be used for scalp, body and pubic lice. It is *inflammable*.

Corn Collodion—See Collodion.

Cresol USP—The Compound Solution of Cresol popularly known as Iycol is a powerful germicide. It may be used for *disinfection* of the skin, lubrication of the hands and as a *vaginal douche* in 1 to 5 per cent aqueous solution.

Crystal Violet—Crystal Violet is one of the rosaniline dyes closely related to gentian violet. It is a mild *local antiseptic* and can be used in the treatment of *superficial fungus infections* especially those due to yeasts. It is applied in 1 to 3 per cent aqueous or alcoholic solution. It is presently used chiefly in the treatment of *burns* in a 1 per cent spray or jelly (base of gum tragacanth).

Cuprex—See Copper Sulfate.

DDT—Dichloro-diphenyl trichlorethane or DDT is an invaluable insecticide developed during the course of the conduct of the War. A practically harmless powder for human beings it becomes a powerful insecticide when mixed with talcum or kerosene. It may be sprayed or applied to the clothes or the body. Thus far its principal usefulness seems to be in the control of lice, mosquitoes and pediculi.

Dakin's Solution—See Chlorine.

Delphinium—See Larkspur.

Dermatol—See Bismuth Subgallate.

Destin Ointment—See Cod Liver Oil.

Diazon—See Sea let Red

Dawson Ointment—See Chrysarob

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Epsom Salts—See Magnesium Sulfate

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For *pruritus vulvae*, an ointment containing 1000 IU (0.1 gm) per cc is generally prescribed. For *senile vaginitis*, suppositories containing 5000 IU (0.5 gm) are given. For *gonorrheal vulvovaginitis* in children, suppositories containing 200 to 2000 IU (0.02 to 0.2 gm) are inserted daily.

Ether USP—Ether is employed locally as a *cleansing agent* in the preparation of the skin for surgical procedure. The usual technique of the operating room calls for scrubbing of the skin with benzine, alcohol and finally ether before the application of the antiseptic solution.

Ethyl Alcoholate—See Benzocaine

Ergol—See Oil of Cloves

Felt—See Pyethrum

Foot Powder—See Talc

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Gentian violet in 4 per cent strength is part of the triple-dye preparation used in *burn therapy*.

Glycerol USP—Glycerine is a *solvent*, a *mollifier* and a *mild astringent*. In the latter capacity it is used in *lozenges* and *rectal suppositories*. As a solvent it is employed in preparing *glycerites*. It is also used in oral preparations as a *sweetening* and *thickening agent*.

Glycerin Suppository USP contains approximately 5 gm of glycerin.

Hydrogen Peroxide USP—The official solution of hydrogen dioxide is of approximately 3 per cent strength. Hydrogen peroxide was formerly used to a great

extent as an antiseptic. It depends for its action on the liberation by the peroxidases present in all tissues of nascent oxygen. While the bubbling of oxygen when peroxide is applied seems very reassuring, the sterilizing action is transient and does not have the more lasting effect of iodine or the protein precipitating action of alcohol. It has a use in the treatment of deep wounds which invite anaerobic infection and as a wet dressing in wounds already infected by anaerobes.

Hydrogen peroxide is of value for its mechanical facilities in cleaning suppurative wounds that have scabs and crusts. It may be employed as an enema for impaction of feces and as a vaginal douche in trichomonal vaginitis. It is also used to bleach hair for cosmetic purposes.

Ichthylol—Ichthylol is a dark thick liquid containing sulfur. It is mildly antiseptic and counterirritant when employed in 5 to 50 per cent ointment. It has no specific properties that would seem to warrant the mess produced by its use.

Ichthammol NF (sulfonated bitumen) is similar to ichthylol. In the form of a 10 per cent ointment in petrolatum it is used in place of ichthylol.

Imadyl—See Acetyl beta methylcholine chloride.

The Iodine Group—Tincture of Iodine U.S.P. is the most useful bactericide in the preparation of the skin for surgical procedures and for the disinfection of wounds. It depends for its action on the antiseptic properties of the iodine which are lasting and the more immediate bactericidal effect of the alcohol. It is also valuable in the treatment of fungus affections of the skin.

The incorporation of the iodine and alcohol insures penetration not obtained with other antiseptics. The alcohol and iodine have a protein precipitating action on the tissues which stimulates tissue reactivity and prevents the further penetration by bacteria. For operative purposes 3.5 or 7 per cent solutions are employed. After drying the iodine is removed by alcohol.

Local sensitivity to iodine is not uncommon. Patients may develop a severe dermatitis at the site of application. Continuous use particularly of the colorless tincture will often lead to a severe dermatitis with desquamation of the skin in large flakes.

Iodine burns are prevented by removing the excess iodine by alcohol. The bottle containing the tincture should be well stoppered so that evaporation and concentration do not occur. It is never good practice to cover up the iodine with a tight fitting bandage which will absorb perspiration or as has occasionally been done with a wet dressing. This brings about penetration of the iodine into deep tissues and nearly always leads to a burn. Iodine should not be applied to the foot if the shoe is to be worn over it.

Some of the preparations containing iodine are the official 7 per cent tincture used full or half strength, the tincture to which a 5 per cent potassium iodide is added and the 4 per cent iodine ointment which is unpleasant to handle and probably no more useful than the simple tincture for deep penetration.

Iodoform and thymol iodide (Aristol) are antiseptic dusting powders containing iodine. They are particularly valuable in the treatment of dirty wounds with abundant slough. These powders may be incorporated in gauze strips used for surgical drains. The chemical is liberated slowly and effectively in the tissues of the wounds. The foreign body particles of gauze act as bridges for the growing connective tissues.

Vioform (iodochlorhydroxyquinoline) a proprietary preparation is at times used as a substitute for iodoform because it is almost odorless. It may be applied as a dusting powder to ulcers, infected wounds or burns. It can be incorporated into ointments as petrolatum in 3 per cent strength. It is also used as a trichomonacide.

The use of iodine crystals in the proportion of 40 gm dissolved in 300 gm of goose grease is an old but still effective remedy in some cases of ringworm of the scalp.

Ipecac—Ipecac is used by mouth as an amebicide.

Kephrine Hydrochloride—Kephrine Hydrochloride is a synthetic chemical which causes local constriction of blood vessels. Its action is similar to epinephrine and although not so powerful it is more enduring. It is used to arrest lo

hemorrhage and produces no systemic side-effects because it is only slightly absorbed. It is supplied as the 5 per cent powder, 3 per cent suppository and in bandages and gauze impregnated with the drug.

Lactic Acid—Lactic Acid is introduced into the vagina for the destruction of *Trichomonas vaginalis*. A douche is prepared containing 4 to 8 cc to a quart of water.

Lanolin USP—Anhydrous lanoline or wool fat is an ointment base. It is useful also to overcome marked dryness of the skin (*emollient*). As it is very tenacious it is usually combined with some other ointment, as petrolatum. Hydrous lanolin or hydrated wool fat contains 27 per cent of water. It is used as an ointment base and absorbs about twice its weight of water. It does not become rancid.

Larkspur (*Delphinium*)—Tincture of larkspur, 10 per cent solution in alcohol, is an efficient remedy for *killing lice* on the scalp. It is used undiluted or with an equal amount of water.

Lassar Peeling Ointment—See Betanaphthol.

Lassar Paste—See Zinc Preparations.

Lead Acetate (Sugar of Lead) USP—Lead Acetate is an *astringent* in lead and opium wash. The use of this preparation is to be condemned. The opium is not absorbed and the lead solution is not only dirty but dangerous because of the possibility of producing *plumbism* (p. 67).

Len galls—See Pyogallol.

Lime Liment—See Carron Oil.

Lime Water—See Calcium.

Lotio Alba—See Stillur.

Lysol—See Cresol.

Magnesium Carbonate USP—Magnesium Carbonate is used externally as a *grit* for dusting powder.

Magnesium Sulfate (Epsom Salts)—The saturated (25 per cent) solution of epsom salts is frequently employed as a *wet dressing* or wash. It is supposed to possess mild local analgesic properties.

Mapharse—See Arsenic.

Mecholyl—See Acetyl-beta-methylcholine Chloride.

Menthyl Salicylate—Menthyl salicylate, salol and quinine salts protect the skin of *photosensitive individuals*. Menthyl salicylate is the most effective of these drugs and is used as a 14 per cent or an 11 per cent lotion.

As a *sunburn preventive* it is included in many prescriptions (p. 3140).

Mercurials—Mercury is a powerful protoplasmic poison.

Bichloride of mercury (1:1000) is principally employed to *sterilize* instruments, silk catheters, hands and the urethral meatus prior to catheterization. At times it is used on the skin as a *wet dressing* in dilutions of 1:2000 in scalp lotions in strengths of 0.1 per cent.

Yellow oxide of mercury (1 per cent ointment) applied to the eyes and eyelids acts as a *mild antiseptic* in the treatment of *blepharitis marginalis*. Ammoniated mercury, which chemically is mercuric ammonium chloride, is embodied in ointments of 1 to 10 per cent strength. Applied to the skin it is an *antiseptic* in pyogenic conditions. It is also used in the treatment of *psoriasis* and *seborrhea*. The official USP ointment contains 10 per cent of ammoniated mercury.

Mild mercurial ointment (Blue Ointment) USP is equivalent to about 30 per cent of metallic mercury. Blue Ointment is used chiefly as a *parasiticide* in the treatment of lice. The stronger mercurial ointment of the USP contains about 50 per cent of metallic mercury. This is employed as an *irritant* in the treatment of *syphilis*.

In *venereal prophylaxis* (p 318) against syphilis for destruction of the spirochetes calomel ointment is preferred This contains 33 per cent of calomel in white petrolatum and hydrous wool fat Mercury ointment does not protect against the gonococcus Two to 4 gm of 33 per cent calomel ointment are rubbed thoroughly into all parts of the penis for five minutes Special attention is paid to the retracted prepuce the frenum and the glans The scrotum is also to be rubbed with the ointment The genitalia are then wrapped in wax paper cellophane or toilet paper and the patient is instructed not to urinate for four or five hours

The *United States Army calomel ointment* is prepared as follows

| | |
|------------------------|-----|
| Mild Mercuric Chloride | 300 |
| Benzoinated Lard | 600 |
| White Wax | 50 |

The *United States Navy calomel ointment* differs slightly in its composition

| | |
|------------------------|-----|
| Mild Mercuric Chloride | 330 |
| Camphor | 20 |
| Phenol | 30 |
| Anhydrous Lanolin | 300 |
| Benzoinated Lard | 200 |
| Beeswax | 30 |

An improved venereal prophylactic that affords considerable protection against syphilis gonorrhea and chancroid has been developed by the U S Army It is dispensed in soft collapsible tubes Part of the ointment is expressed into the urethra while the remainder is anointed on the genitals The formula is undernoted

| | |
|-------------------|-----|
| Sulfathiazole | 150 |
| Calomel | 300 |
| Petrolatum | 400 |
| Light Mineral Oil | 140 |
| Cetyl Alcohol | 10 |

Among the *newer derivatives of mercury* of some value as antiseptics are mercurochrome metaphen and merthiolate They have strong antiseptic action and very low toxicity It was hoped that merthiolate might be used successfully for intravenous administration in cases of sepsis and subacute bacterial endocarditis This hope however has not been realized in actual practice

Cinnabar (red sulfide of mercury) acts as an antiseptic It is used in ointments or shake lotions in concentrations of 1 to 10 per cent In recent years it has been applied by tattooing in the treatment of intractable pruritus ani with considerable success in some cases

Acid Nitrate of Mercury is a strong *corrosive* used by some dermatologists in the treatment of basal cell epithelioma It is a powerful drug and should be applied with great caution

Menthol—See Volatile Oils.

Methanamine—See Formaldehyde

Methyl Salicylate (Oil of Wintergreen) USP—Oil of wintergreen is a *counterirritant* and *rubefacient* when applied to the skin It is also mildly antiseptic

Milk—Milk is the simplest and most easily available protective solution for the wet dressing It makes a very soothing dressing when applied cold in acute dermatitis

Mustard—Mustard is a *counterirritant* and *rubefacient* as is the mustard paste or mustard plaster

The popular *mustard foot bath* causes a profuse diaphoresis It is prepared by adding a half can of commercial English mustard to a pail of warm water The feet are immersed until a local rubefacient action occurs The diaphoresis is aided by warm wrappings to the body and the sipping of hot lemonade spiked with whiskey

The official *Mustard Plaster USP* may be purchased Before applying the commercial mustard plaster it is thoroughly moistened with tepid water and then

placed on the skin. The skin is watched for the rubefacient effect. Blistering should not be produced.

A *domestic mustard poultice* may be prepared by mixing one part of mustard to two to three parts of flour. Warm water is added to form a paste. This poultice is applied until a local blush appears.

Myrrh USP—Tincture of Myrrh USP is used as an *astringent mouthwash* and a *gargle*.

Nitric Acid USP—Nitric Acid, a powerful protoplasmic poison, is used to *cauterize* wounds made by possibly rabid animals. It is also employed for the removal of small epithelial growths such as *warts* and *nevi*.

The technique of nitric acid cauterization requires that the surrounding healthy tissue be protected with petrolatum. The nitric acid is then applied on the end of a glass rod.

Nupercaline—See Benzocaine.

Oil of Cloves USP (See *Volatile Oils*)—Oil of Cloves is *antiseptic* and *aromatic*. It is used in dental practice as a *local anodyne* when placed on a piece of cotton and inserted into the cavity of a carious tooth. A derivative of oil of cloves, *eugenol*, is at times preferred because of its greater stability. Many dentists make a stiff paste of eugenol and zinc oxide which they pack into painful carious teeth. This mixture sets and is retained as a cast.

Oil of Cinnamon—Oil of Cinnamon is an *aromatic*, usually prescribed as the official *Cinnamon Water USP*.

Oil of Eucalyptus USP—Oil of Eucalyptus is *antiseptic* and *expectorant*. It may be added to the tea kettle for *soothing inhalation*. It is used in sprays in 10 per cent concentration for treating inflammations of the nasal mucous membrane.

Oil of Theobroma (Cocoa Butter) USP—Oil of Theobroma is used in pharmacy for making *suppositories* and *massage oils*. It may also be applied to sore nipples.

Oil of Turpentine USP—Rectified Oil of Turpentine is applied externally as a *rubefacient* and *counterirritant*. It is commonly applied with abdominal *stupes* for the relief of distention.

Orthoform—See Benzocaine.

Oxyquinoline Sulfate—Oxyquinoline Sulfate is a strong antiseptic used in many *opolyactic douches* and *contraceptive jellies*. It is present in a proprietary ointment (*Q inolor*) in combination with benzoyl peroxide and essential oils. This ointment is of considerable value in the treatment of refractory *sycondysia vulgaris*.

Pectin—Pectin pastes are useful *protectives* and *vehicles* in *ulcer therapy*. Tragacanth (p 3130) sulfonamides (p 3138) ethylamino benzoate (p 3144) and/or zinc peroxide may be added for specific effects.

Thin Pectin Paste

| | |
|-----------------------------|-------------|
| Benzoic Acid | 20 |
| Pectin Citrus No. 100 Grade | 600 |
| Glycerin | 600 |
| Ringer's Solution | qs ad 10000 |

Dense Pectin Paste

| | |
|-----------------------------|-------------|
| Benzoic Acid | 20 |
| Pectin Citrus No. 100 Grade | 1500 |
| Glycerin | 1500 |
| Ringer's Solution | qs ad 10000 |

Fill the ulcer crater with the paste, cover with a double thickness of cellophane slightly larger than the ulcer and fasten down with 2 inch strips of adhesive tape. If the patient is restless or the skin is too sensitive for the use of the tape, cover the cellophane with a binder. To prevent tearing of the cellophane, arrange the

patient's position so that he does not lie on the ulcer. If this happens and some of the paste drains out slip in fresh paste. Do not change dressings oftener than once a day unless the wound is contaminated with excreta. In some cases the dressing does not need changing for two or three days. The progress of the ulcer may be watched without disturbance through this nonirritative waterproof covering of cellophane.

Penicillin—Local and topical application of penicillin affords great promise for the relief of a variety of dermatoses. Penicillin may be applied in physiological saline solution made so that 1 cc equals 250 to 1 000 units. It can be unprovoked in an ointment base of 50 per cent hydrous wool fat and 50 per cent rose water ointment so that each 30 gm contains 100 000 units or approximately 3300 units to the cc. The preparation should be stored in the icebox and probably loses its potency within a few weeks. Amongst the conditions benefited include pyoderms, streptococcal infections, impetigo contagiosa, folliculitis, carbuncles and syccosis barbae. Penicillin unfortunately is of no value in acne vulgaris unless there is secondary infection.

Petrolatum (Petroleum Jelly) USP—Petrolatum is employed externally and internally as a lubricant.

The White Petroleum Jelly (White Petrolatum) USP is a lubricant and an ointment base. This simple ointment base contains 90 per cent white petrolatum and 5 per cent each of wool fat and white wax.

Liquid Paraffin or Liquid Petrolatum USP is the white mineral oil employed externally in atomizers for spray purposes and also to soften and protect dry skins. Its use in nasal and throat sprays may lead to the production of lipid pneumonia (p 2048). Internally the heavy liquid petrolatum is a lubricant in the treatment of constipation. It is also useful in cleaning ointments from the skin.

The emulsion of liquid petrolatum gives a lubricant laxative action. Melted paraffin may be sprayed on denuded skin surfaces as a mechanical protective.

The Phenol Group—Phenol is a general protoplasmic poison. The intense irritative and escharotic action of strong phenol limits its use in clinical medicine. Idiosyncrasy occasionally causes renal irritation. This is especially true when phenol is in great dilution is applied over wide areas following greater absorption.

Phenol is used in 5 per cent solution for disinfecting instruments. When dissolved in oils or ointments in 1 per cent strength its effect is not only on bacteria but also on tissues themselves where it acts as a protein precipitant, an irritant and a mild antiseptic.

It may be employed in full strength as a corrosive and antiseptic to cauterize snake bites. It is of no important value as a disinfectant gargle. It has frequently been taken internally as a poison, the treatment of which is discussed in the section on Toxicology (p 757).

Pure phenol may be used on a toothpick for the painless drainage of small suppurations of the skin. In 0.25 to 1 per cent solution added to a spray lotion or ointment phenol is antipruritic. Phenol should never be applied to a single area for any length of time especially as a wet dressing as it may produce a burn and even lead to gangrene.

Derivatives of phenol are also used as bactericides. Cresol and tri-cresol are of very much stronger bactericidal substances than phenol but are rarely used in therapeutics.

Picric Acid or Trinitrophenol is an effective antiseptic which is frequently used for disinfecting the skin prior to surgical operations. It may replace iodine in the patient who is sensitive to the latter substance. Patients however will frequently develop dermatitis after contact with picric acid. By virtue of its protein precipitating and mild local anesthetic action picric acid and its derivatives are also of value in the treatment of burns. Suppositories of silver picrate are used in the treatment of *Trichomonas vaginalis* infections.

Resorcinol is usually incorporated in scalp lotions and ointments because of its irritant and rubefacient effect as well as its mild antiseptic action. Resorcinol will discolor gray or white hair under which circumstances resorcinol monacetate may be substituted (see also Resorcinol).

Thymol a derivative of phenol is a useful fungicide. It is employed in dermatophytosis in the following formula

| | |
|-----------------------|-------------|
| Thymol | 1.0 |
| Salicylic Acid | 5.0 |
| Alcohol (70 per cent) | qs ad 100.0 |

The oral and local use of thymol has proved effective in some cases of actinomycosis. Thymol is a flavor in many mouth washes. Taken internally it is a useful anthelmintic (p 1891). Thymol iodide (Aristol) of the U.S.I. is a valuable antiseptic dusting powder which liberates free iodine and has some bactericidal activity.

Phenylmercuric Nitrate—Phenylmercuric Nitrate is a fungicide used in ointments in 1:1000 strength for treatment of dermatophytosis.

Phenyl Salicylate (Salol)—Salol is used externally as a protective in photosensitive individuals especially those with lupus erythematosus. It may be incorporated in lotions or creams in a strength of 2 per cent.

A representative formula follows

| | |
|-------------------|-------------|
| Phenyl Salicylate | 2.5 |
| Zinc Oxide | |
| Talc | aa 20.0 |
| Glycerin | 15.0 |
| Water | qs ad 120.0 |

Percic Acid (Trinitrophenol) USP—See Phenol Group

Potassium Permanganate USP—Potassium Permanganate is a powerful oxidizing agent. Explosions are liable to occur when it is brought into contact with organic or other readily oxidizable substances either in solution or in the dry condition.

Externally potassium permanganate is deodorant, irritant, astringent and mildly germicidal. On the mucous membrane it may be employed in dilutions of 1:5000 to 1:1000. It serves well as a wet dressing or bath for acutely inflamed moist eczematous areas in aqueous solutions of 1:5000. It stains tissues a mahogany brown and also discolors the fingernails and toenails.

The potassium permanganate bath may be used in the treatment of crusty scaling generalized eruptions that have a foul odor as pemphigus. Approximately 5 to 50 gm. of the crystals are dissolved in the tub. Both patient and tub are stained deeply so that this type of procedure should be reserved only for more or less generalized eruptions.

Protargol—See Silver

Pyrethrum—Pyrethrum an extract obtained from a certain type of chrysanthemum blossom is a powerful parasiticide and insecticide. It is the active component of many commercial insecticides such as Flit. It has been recommended (and is available as a proprietary preparation) for the treatment of scabies. It is supplied as an ointment containing 27 per cent of the drug. The tendency for pyrethrum to produce dermatitis would make other scabecides preferable.

Pyrogallol (Pyrogallie Acid)—Pyrogallol a phenol derivative is an irritant and antiseptic substance. In strong concentrations it has a corrosive action upon the skin. Its use is generally restricted to the treatment of certain localized cases of lupus vulgaris where it is applied as an ointment in 5 to 25 per cent strength on a dressing. The dressing is changed daily. The drug produces a superficial slough which eventually separates.

A commercial derivative Len gollol is used in 5 to 10 per cent strength in zinc oxide ointment in the treatment of chronic eczema and psoriasis.

Quinine—Quinine sulfate and bisulfate are used as light protectives in photosensitive persons. They may be incorporated in creams, lotions or face powders usually in strengths of 2 to 4 per cent. Typical formulas will be found in the section on cosmetics devoted to the discussion of sunburn preventives (p 3140).

Quinolor—See *Oxyquinol no Sulfate*

Radium—Radium usually available as one of the salts of the element is of chief value in the treatment of accessible *malignant tumors* of the skin and mucous membranes. It is also used in some of the larger *cavernous hemangiomas* of the skin. Its use is fraught with great danger to both patient and physician *except in the hands of the expert*. Radiotherapy is more fully discussed elsewhere (p. 3197).

Resorcinol U S P—Resorcinol is *antiseptic antipruritic locally astringent and keratolytic*. Dilutions of 1 to 3 per cent applied externally are astringent and antipruritic. The higher concentrations of 10 to 20 per cent particularly in ointment form produce *exfoliation* and are likely to prove very irritating.

Resorcinol is used in *hair tonics* where it is of value in seborrheal conditions. Resorcinol stains blonde gray and white hair a dirty yellow or greenish color. It is preferable to use resorcinol monoacetate which does not discolor hair.

R Compound Resorcin Lotion

| | |
|-----------------------|-------------|
| Bichloride of Mercury | 0.12 |
| Resorcinol | 60 |
| Alcohol (70 per cent) | 600 |
| Distilled Water | q.s ad 1200 |

R Compound Resorcin Ointment

| | |
|---------------------|------------|
| Salicylic Acid | 0.90 |
| Precipitated Sulfur | 0.90 |
| Resorcinol | 0.90 |
| Petrolatum | q.s ad 300 |

Resorcinol is the principal ingredient of Castellani's paint, a valuable fungicide. The prescription is compounded by adding 10 cc of saturated alcoholic solution of basic fuchsin to 100 cc of 5 per cent phenol in aqueous solution. After filtration 10 cc of boric acid is added and the mixture stands for two hours at the end of which time 5 cc of acetone are added. After another two hours wait the final product is completed by the addition of 10.0 cc of resorcinol. Castellani's paint is stored in a dark stoppered bottle.

Rotenone—Rotenone is a proprietary preparation, a derivative of derris root which has long been used as an *insecticide* in agriculture. It is obtainable commercially for medicinal use as a 2 per cent emulsion which does not stain the bedding or underwear. It is particularly recommended for the treatment of *scabies*.

Rose Water Ointment—See *Cold Cream*

Salicylic Acid U S P—Used externally. Salicylic Acid is a mild *antiseptic* and a *keratolytic*. It is also employed as an *astringent* and *fungicide*. The pure crystals of salicylic acid may be employed in 2 to 5 per cent concentration in a *dusting powder* employing starch or talcum as the vehicle. In 1 to 2 per cent alcoholic solution salicylic acid acts as an *astringent*. The popular Whitfield's ointment is essentially 6 per cent salicylic acid and is employed in the treatment of dermatophytosis.

R Whitfield's Ointment—full strength (Ointment of Benzoic Acid and Salicylic Acid N.F.)

| | |
|----------------|---------------|
| Salicylic Acid | 18 |
| Benzoic Acid | 36 |
| Lanolin | |
| Petrolatum | aa q.s ad 300 |

As a stronger *keratolytic* 20 per cent salicylic acid in the official Corn Colloid ointment is widely employed as a discutient.

Salol—See *Phenyl Salicylate*

Sal arsan—See *Arsenic*.

Scarlet Red (Scarlet R)—Scarlet Red is an azo dye combined with betanaphthol. It is generally used in the form of an ointment containing 4 to 8 per cent of the active ingredient. A commercial preparation Dimazon is prepared as an ointment containing 2 per cent of the dye and 98 per cent of petrolatum.

Scarlet Red has been recommended as a stimulant to epithelial growth in the treatment of burns, wounds and chronic ulcers. Other cheaper and more effective preparations are available for the purpose.

Silver—Silver nitrate is a powerful protoplasmic poison. Weak solutions are astringent; stronger solutions are antiseptic and powerfully germicidal.

The modern employment of silver nitrate is almost wholly limited to its caustic action. In the form of Lunar Caustic USP, the silver pencil is employed to burn down exuberant granulations, to touch up and stimulate a local fissure or excoriation or as a local hemostatic as in epistaxis where the bleeding point is readily accessible. Its repeated use on erosions, fissures and ulcers in the mouth may stimulate excessive epithelial hyperplasia and lead to malignant change.

In the prevention of gonorrheal conjunctivitis in the newborn, one drop of 2 per cent silver nitrate solution is instilled into each conjunctival sac immediately after delivery. Under any other circumstances, the silver should not be used in strengths beyond 0.2 to 2 per cent in the conjunctival sac. Stronger solutions may cause corneal destruction and scarring leading to blindness.

Silver nitrate wet dressings 0.125 to 1 per cent strength are useful in infected and impetiginized eczemas. For urethral instillation in the treatment of gonorrhea, the first inject on should be no stronger than 1:10,000 and the strength may then be increased to 1:2,000. It has little place in the modern treatment of gonorrhea. The irritation from the silver may be so intense as to produce a chemical urethritis. It is quite possible that many instances of nonspecific urethritis, often treated as gonorrheal urethritis, result from the overenthusiastic use of the silver solution as a venereal prophylactic.

The organic silver preparations Protargol and Silvol have largely replaced organic silver nitrate. Protargol is an organic silver preparation of approximately 8 per cent strength. Silvol (Argyrol) is a colloidal silver proteinate containing 19 to 25 per cent of silver.

The solutions of the organic silver preparations must be freshly prepared with distilled water. They should be dispensed in amber colored bottles that are to be protected from light.

While the organic silver preparations are as powerful as the silver salt, they neither precipitate protein nor cause significant irritation. They may be used in saturated aqueous form on a tampon employed intranasally or intravaginally. The former procedure is widely employed in the treatment of the upper respiratory infections, though it would seem to be of little practical value.

Protargol (2 per cent) and Silvol (25 per cent) may be employed as a venereal prophylactic against gonorrhea. The solution should be instilled into the urethra immediately after exposure and held for five to ten minutes. This measure has no efficacy as a spirocheticidal agency.

Protargol (0.5 per cent) or Silvol (10 to 25 per cent) are used in the conjunctival sac and Protargol may be employed for urethral irrigation in 1:1,000 to 1:2,000 solution.

The long continued use of the silver preparations on mucosal surfaces is inadvisable. Absorption of the silver may lead to production of argyria (p. 136).

Soap—The soap of the USP is Castile Soap prepared from sodium hydroxide and olive oil. The Soap Liniment USP contains mild soap, camphor and oil of rosemary.

Soft soap (green soap) is prepared from linseed oil, potassium hydroxide, sodium hydroxide, glycerin and water. It is conveniently dispensed as the Tincture of Green Soap USP, which is an alcoholic solution containing 65 per cent of soft soap perfumed with oil of lavender. The soft soap may be used as a vehicle for ointments and as a detergent.

Sodium Bicarbonate USP—Sodium Bicarbonate is used externally as an antacid mouthwash or as an antipruritic. It may be applied to the skin in the form of

paste when mixed with water. It is useful in the treatment of *generalized pruritus* using 1 pound in the tub of lukewarm water.

Sodium Borate (Borax) USP—Sodium Borate a mild antiseptic and astringent is used in 1 to 2 per cent solution as a *collyrium*.

Sodium Perborate USP—Sodium Perborate is an oxidizing and germicidal agent. Its principal use is as a mouthwash in the treatment of *Vincent's infection* (*fusospirochilosis*) of the gums or other soft tissues of the mouth or pharynx. It should be diluted as 1 level teaspoonful to a tumbler of water or in about 2 per cent solution. Protracted usage is undesirable as it may lead to *chemical stomatitis*.

The powder may be dusted upon infected especially anaerobic wounds of the skin.

Sodium Propionate—Sodium Propionate is a potent fungistat with low toxicity used in the treatment of *dermatomycoses*. It is commercially available as a sopronol solution, ointment or powder and appears to be effectual and nonirritating for prophylactic and curative uses.

Sodium Thiosulfate (Sodium Hydrosulfite) USP—Thiosulfate in the form of a lotion is a *paranticide* and *fungicide*. The strength of the application may be a 10 per cent aqueous solution or a 12 per cent ointment. It is particularly useful in the treatment of *tinea versicolor*.

Spanish Flies—See *Cantharides*.

Starch USP—Starch (corn) or *Amylum* is employed topically as a *dusting* and *drying powder* as well as a *diluent* for more active powdered substances.

The starch bath is of great value as a *protective* and *antipruritic*. It may be prepared with bran or cornstarch. On half to one pound of the bran is placed in a gauze bag. The bag is saturated with very hot water after which it is placed in the tub and squeezed into the water or used over the body as a wash cloth.

The cornstarch bath is prepared by adding one half to one pound of a popular brand of laundry starch such as Lanit or Argo directly to the tub.

Sulfanilamide, Sulfathiazole and Sulfadiazine—Much of the initial enthusiasm for topical applications of sulfonamide has dissipated. The introduction into wounds of crystals, powders, pastes and aqueous solutions for prophylactic and curative treatment has for the most part been abandoned. Local healing is delayed and serious idiosyncrasy may be experienced. The advantages which may accrue are more likely due to systemic absorption better produced by oral or parenteral introduction far from the site of the lesion.

There is much to commend the local application of sulfonamide ointment to the unbroken skin particularly in the pyoderms. Under these circumstances an ointment of 5 per cent sulfathiazole is recommended. For spray treatment in the management of *burns* and *nasopharyngeal disorders* 3 per cent sulfadiazine in 8 per cent triethanolamine has been recommended but the results have not substantiated the hopes of the innovators.

Sulfur—Sulfur is used externally as a *parasiticide* particularly against *scabies* and in the treatment of *seborrheal conditions*. It is also useful in *fungus infections* and some other skin diseases.

The official *Sulfur Ointment* contains 15 per cent of precipitated sulfur. When applied to the face it is *diluted at least five times*. It should not be applied directly to the eyelids. Sulfur is also used as a powder or it may be incorporated in face powder.

Sulfur baths popularly employed at many of the spas are the equivalent of 50 to 150 gm. of crude potassium sulfate dissolved in a tubful of water. It is almost a certainty that no absorption or constitutional effect is demonstrable.

The popular *Hemlock's lotion* (*Liquor Calcis Sulfuratae NF*) is employed as a *keratolytic* particularly in the treatment of *indurated acne*. It is applied diluted as a compress as 4 to 15 cc. in a pint of hot water. It stains fingernail and tarnishes silverware.

R For Scabies

| | |
|---------------------|-----------|
| Precipitated Sulfur | |
| Balsam of Peru | aa 20 |
| Benzoinated Lard | qs ad 300 |

R Wilkinson's Ointment (Compound Sulfur Ointment NF)

| | |
|--------------------------------|-----|
| Sublimed Sulfur | 150 |
| Oil of Cade | 150 |
| Precipitated Calcium Carbonate | 100 |
| Soft Soap | 300 |
| Solid Petroxoline | 300 |

R For Acne

| | |
|---------------------------------|-------|
| Potassium Sulfurate | 4-120 |
| Zinc Sulfate | 4-120 |
| Distilled Water | 1200 |
| Sig Lotio Alba for external use | |

R (Dane's)

| | |
|-------------------------------|--------|
| Precipitated Sulfur | 60 |
| Resorcin | 20 |
| Sulfonated Bitumen (Ichthvol) | 100 |
| Zinc Oxide | |
| Starch | aa 160 |
| Petrolatum | |
| Lanolin | aa 200 |

R For Seborrheic Dermatitis

| | |
|---------------------|-----------|
| Salicylic Acid | 06 |
| Precipitated Sulfur | 06 |
| Cold Cream USP | qs ad 300 |

R Precipitated Sulfur

| | |
|------------------|-----------|
| Salicylic Acid | |
| Oil of Cade | aa 18 |
| Benzoinated Lard | qs ad 600 |

Talc Powder USP—Purified Talc is nonscented hydrous magnesium silicate. It is used on the skin for protection from the air from friction in intertriginous areas and also as an absorbent. Active ingredients may be incorporated in it such as menthol, boric acid, thymol or salicylic acid. It is also used in the preparation of shake mixtures and pastes (F).

The United States Army Foot Powder is prepared as follows:

| | |
|------------------|-----|
| R Salicylic Acid | 20 |
| Boric Acid | 60 |
| Zinc Stearate | 30 |
| Exsiccated Alum | 10 |
| Starch | 100 |
| Powdered Talc | 780 |
| Make a powder | |

Tannic Acid USP—Tannic Acid is employed as an astringent, antiseptic and hemostatic. Two and one-half to 5 per cent solutions may be sprayed on burns; the result is coagulum acting as a protective dressing and preventing the excessive loss of body fluids.

As an astringent bath, 7 to 10 ounces of tannic acid may be added to the usual bath tub. A 20 per cent ointment or a suppository containing 0.1 gm is useful in the treatment of hemorrhoids.

The official USP Glycerite of Tannic Acid is a 20 per cent solution in glycerin. For local application the glycerite should be diluted to make the tannic acid concentration between 0.5 and 2 per cent.

| | |
|-----------------------|------------|
| <i>Is an Androtic</i> | |
| I, Tannic Acid | 50 |
| Alcohol | 1000 |
| Water | qs ad 1000 |

Tar—A number of tars are used therapeutically. Their effect is irritant, keratoplastic and antipruritic. They are especially valuable in chronic eczemas.

Coal tar or *Lix Carbonis N.F.* obtained by destructive distillation of coal is used in ointments in 2 to 10 per cent strength. A derivative *Liquor Carbonis Detergens N.F.* is weaker and may be used in ointments or lotions in strengths of 3 to 20 per cent. The tar bath is prepared by adding 100 cc. of *Liquor Carbonis Detergens* to the tub.

Juniper Tar or Oil of Cade *U.S.P.* is used in ointments in dilutions of 1 to 10 per cent. Pine tar or *Lix Liquida U.S.P.* is used in 2 to 10 per cent strength in ointments. The official Pine Tar Ointment *U.S.I.* contains 50 per cent of pine tar. Rectified Oil of Birch Tar or *Oleum Rusci N.F.* is applied in 2 to 10 per cent strength.

Treatments with tar are begun with a weak strength which may be gradually increased if no untoward reaction (*dermatitis*) occurs. Tars are avoided in hairy parts because of a tendency to cause *pustular folliculitis*.

Several colorless or white tar preparations are available in proprietary preparations where the disagreeable color must be avoided (*Taralba Supertah Pixaboi*).

Coal Tar Ointment

| | |
|-------------------|-----|
| I, Crude Coal Tar | 40 |
| Zinc Oxide | 40 |
| Castor Oil | 40 |
| Cornstarch | 300 |
| Petrolatum | 260 |
| Make an ointment | |

Thymol—See Phenol Group

Tragacanth *U.S.P.*—Tragacanth forms a mucilage on addition of water. It is used in the pharmacy to make emulsions. A wider application is in *greaseless lubricants* that are particularly used for catheters, contraceptive jellies and as applications to the chapped skin.

| | |
|------------------|------------|
| I, Tragacanth | 40 |
| Glycerin | 600 |
| Boric Acid | 100 |
| Water | qs ad 5000 |
| Oil of Bergamot | 06 |
| Sig. Hand Lotion | |

The official Mucilage of Tragacanth may be used as a *demulcent* base for other medications to be applied to the skin.

| | |
|---|-------------|
| I, Mucilage of Tragacanth <i>U.S.P.</i> | |
| Tragacanth | 60 |
| Glycerin | 180 |
| Distilled Water | qs ad 1000 |
| I, Tragacanth Paste | |
| Benzoin Acid | 20 |
| Glycerin | 1000 |
| Tragacanth Powder | 1000 |
| Ringer's Solution | qs ad 10000 |

Trichloroacetic Acid *U.S.P.*—Trichloroacetic Acid is usually prepared in saturated aqueous solution. It is used as a caustic and local hemostatic. Its principal application is for the removal of *xanthelasma palpebrarum* (p. 3211) but it is usually

is used to flatten out *mollusca* warts. The surrounding healthy skin is protected with petrolatum.

Triethanolamine Technical NNR—Triethanolamine is an excellent *emulsifying agent* used in the preparation of dermatologic preparations. It is said to *increase the penetrating power of oily materials* and to have some *bacteriostatic action*. Combinations with fatty acids form soaps with good detergent properties. Emulsions are prepared by dissolving the fatty acids in oil, dissolving the triethanolamine in water and by finally mixing these two solutions. Emulsions are prepared in strengths of 20 to 40 per cent which may later be diluted.

Tyrosine—Tyrosine, a derivative of gramicin, is a powerful antiseptic agent that is available in 2 per cent solution. Diluted with sterile distilled water so that 1 cc. contains 33 mg. it may be used as a wet dressing in infections of the skin. Absorption and parenteral administrations may be followed by destruction of erythrocytes and toxic symptoms.

Undecylenic Acid—Undecylenic acid is an antimicrobial oil under the trade name of D. Senex.

Vanishing Cream U.S.P.—Vanishing creams are emulsions of oil in water and consist chiefly of a soap emulsifying agent (potassium hydroxide), free stearic acid and water. To these are added other substances such as glycerin, mineral oil and cocoa butter. Triethanolamine may be used as the emulsifying agent. Such creams are not so greasy or protective as ordinary ointments and are absorbed less completely by the skin. In dermatology they are used principally as vehicles where an occlusive greasy substance is undesirable.

| | |
|--|------|
| Ib Stearic Acid | 12.5 |
| Glycerin | 6.5 |
| Distilled Water | 7.0 |
| Potassium Carbonate | 1.2 |
| Sig. Vanishing Cream (see also E. p. 3136) | |

Vaccineal Prophylactics—See Mercapto.

Vioform—See Iodine Group.

Vitamin K Solution—See Calcium.

Volatile Oils—The volatile oils include camphor, menthol and wintergreen. For external use they are employed in weak solutions to give odor and taste. Thus Camphor Water U.S.P. is widely employed as a vehicle for collirs. Camphor in strengths of 0.125 to 1 per cent may be added to aqueous or alcoholic solutions, lotions, ointments, etc. for its antipruritic effect. The Liniment of Camphor (Camphorated Oil) U.S.P. is employed as a counterirritant and rubefacient.

Menthol U.S.P. 0.5 to 2 per cent in powder, oily solution, shake lotion or ointment, may be used as an antipruritic and cooling agent. It is avoided where there are open skin lesions for it produces an intense burning sensation. Menthol is commonly used in nose and throat sprays and drops in approximately 1 per cent solution as a mild antiseptic and stimulant. Solid menthol may be employed for inhalation in rhinitis. It is the active ingredient of most headache remedies that are locally employed. The beneficial effect is due to the cooling and counterirritation.

Oil of Wintergreen is applied locally for relief of rheumatic pains.

Whitfield's Ointment—See Benzoic Acid.

Wilkinson's Ointment—See Sulfur.

Wintergreen—See Methyl Salicylate.

Xerofom—See Benzoic Tribromphenol.

Xylene—Xylene destroys lice and penetrates and destroys their ova. It may be used with equal parts of alcohol and ether.

If the skin is sensitive and excoriated use

| | | |
|---|------------|-----------|
| R | Xylene | 40 |
| | Petrolatum | qs ad 300 |

Zinc Preparations—Zinc Acetate USP is a mild astringent and antiseptic. It is commonly used in *collyria* in the strength of 0.1 to 0.5 per cent.

Zinc Oxide is slightly antiseptic and feebly astringent. It may be used as a *dusting powder*. The Zinc Oxide Ointment USP is 20 per cent strength. Prepared calamine is essentially zinc oxide with a little ferric oxide.

Zinc Sulfate is employed chiefly in ophthalmology. It is used as a wash in 0.1 to 1 per cent dilution particularly in the *conjunctivitis* caused by the *Morax Axenfeld bacillus*.

Zinc peroxide is used as a paste in the treatment of certain *micro-aerophilic streptococcal* infections and ulcerations of the skin.

| | | |
|---|---------------------|----------------------------|
| R | Zinc Peroxide Paste | |
| | Zinc Peroxide | 100 |
| | Distilled Water | sufficient to make a paste |

This paste has given excellent results in undermined and spreading ulcers caused by *micro aerophilic streptococci*.

Zinc stearate may be used as a *dusting powder*.

Zinc salts are important components of several widely used *shale lotions and pastes*.

| | | |
|---|-------------------------------|------------|
| R | Compound Starch Lotion (Unna) | |
| | Zinc Oxide | |
| | Talc | ss 200 |
| | Glycerin | 150 |
| | Water | qs ad 1200 |

| | | |
|---|--|-----------|
| R | Lassar's Paste (Paste of Zinc Oxide A F) | |
| | Zinc Oxide | |
| | Starch | ss 75 |
| | Petrolatum | qs ad 300 |

To this paste may be added active ingredients such as salicylic acid.

METHODS OF APPLYING LOCAL OR TOPICAL MEDICATION TO THE TEGUMENTARY SYSTEM AND THE ACCESSIBLE MUCOUS SURFACES

The body surfaces are accessible to treatment by watery or alcoholic solutions, by fat soluble preparations and by insoluble materials. The methods of application depend upon the physical properties of the drug and the therapeutic aim.

AQUEOUS AND/OR ALCOHOLIC PREPARATIONS

Watery and/or alcoholic solutions applied to the unbroken skin produce their most marked effects upon the *epidermal* structures. They have relatively slight effect on the *derm* corium. Water soluble preparations are used for the general or local *ablution* or the *wet dressing*. Only limited amounts of alcoholic preparations can be applied or dabbed on smaller areas of skin surfaces.

The solutions are employed as *baths*, *wet dressings* or *washes*.

A. Baths—As therapeutic agencies, baths are medicated to afford *protection*, *relieve itching*, *effect counterirritation* or to produce *maceration*.

The Cleansing and Deodorant Bath—The temperature of the average cleansing bath approximates 100° F. Many women take a tub bath at a considerably higher tem-

perature Such baths are often stimulating Taken at night they may provoke *insomnia*

In the ordinary *tub bath* the body is washed with a soft cloth and/or soap The extremities and hairy parts are scrubbed with a brush After the tub bath the skin is cleansed by repeated rinsings using a warm spray or preferably the cold or cool shower It is unwise to go out into the open air for at least an hour after a tub bath A modern substitute for the tub bath is the *shower* The water preferably should be hot or warm in the beginning The patient liberally lathers and soaks the body particularly the hairy parts and regions of the body orifices The shower is terminated by a cool or cold washoff depending upon the reaction of the skin After the bath the skin is dried by patting rather than by excessively vigorous rubbing There is no danger in going outdoors following the shower The shower is far more hygienic than the tub The patient does not wallow in his own dirtied water

Those who have adequate bathroom facilities should be encouraged to take a *daily tub or shower* This is particularly pertinent to adolescents and younger children whose skins are apt freely to secrete sweat and oil A daily bath for older people may cause or aggravate dryness of the skin and so lead to *senile pruritus* (p 314) So called winter itch may actually arise from or be made worse by excessive bathing Many dermatologists suggest the use of the *shower cap* It is their belief that the hair should be washed and soaped no more than once or twice a week lest the scalp become excessively dry and laden with dandruff

Children preferably are bathed at night immediately before retiring For adults the shower is best taken in the morning the tub bath in the late afternoon or at bedtime

For *bath soap* the inexpensive nonscented noncolored preparations are preferred Highly *alkaline and hard water soaps* cause considerable skin irritation The *expensive soaps* that are colored and scented may give rise to contact dermatitis

Soap substitutes are obtainable in the form of various *sulfonated oils or higher alcohols* These include *Tersus* (Doak Co) and *Acisolate* (National Oil Products Co) These are useful in the dry or eczematized skin Their effects must be watched since they may induce further irritation

Protective Soothing Antipruritic Bath (Colloidal Bath)—A protective soothing and antipruritic bath is obtained by adding to the tub 1 pound of a refined soluble cornstarch such as *Lu* or *Argo* or an equal amount of bran enclosed in a gauze bag with $\frac{1}{4}$ pound of baking soda The starch is made into a thin paste with cold water and stirred well before placing in the tub

Patients with rough dry skins may add $\frac{1}{4}$ to 1 ounce of mineral oil to the tub An exceedingly hot bath will have an antipruritic effect without added medicament

Counterirritant or Rubefacient Bath—A counterirritant or rubefacient effect is obtained from the hot unmedicated bath The reaction may be enhanced by the addition of mustard The full mustard bath is prepared by mixing English mustard flour and tepid water in a small basin The paste is added to the warm tub The patient must not rub the mustard water near the eye

Macerating and Keratolytic Baths (Deodorant and Antiseptic)—With exudative inflammation of the skin crusting and scaling causes discomfort and an unpleasant odor The removal of the offensive materials is accomplished by a bath of *potassium permanganate* Dissolve 1 teaspoonful to 1 tablespoonful of the crystals in a quart of water After the crystals are completely dissolved add the solution to the tub This is useful in *secondarily infected lesions* generalized superficial *pyoderma* and in extensive crusted and oozing dermatoses The potassium permanganate solution stains the skin and tub Care must be taken that the crystals are completely dissolved as their contact with the skin will produce a chemical burn

Antipruritic and Anodyne Baths—In the treatment of *generalized pruritus* the full bath is exceedingly useful The hot bath without medication and the bran or cornstarch baths soothe and allay the discomfort If these measures are not successful and the itching is severe tar baths are tried Three ounces of *Liquor Carbonis Detergens* or the *Almay Tar Bath* preparations are added to the tub Baths of these types tend to reduce *excessive perspiration* particularly if followed by an alcohol rub and a liberal use of talcum powder

Healing Baths—Sulfur baths are popularly used at many of the spas. They are employed for their alleged deep effect on the skeletal system and for their efficacy in generalized dermatoses. Sulfur baths are accomplished in the home by adding 50 to 150 gm of crude potassium sulfate to the tub. This is a foul smelling mixture which stains the tub. It is not to be suggested for routine use in the home since the efficacy of the measure is questionable to say the least. Baths with *Vlemmckx's solution* (solution of sulfured lime) 150 cc to the tub are valuable in treating widespread pyogenic infections particularly *pustular folliculitis* in hirsute individuals.

Contraindications to Bathing—As with all other useful therapeutic measures the bath has untoward effects. Most skin eruptions of the *eczematous types* particularly those with acute inflammation and vesiculation are made significantly more intense as the result of the application of water.

Debilitated and *elderly* patients may become faint and enervated following a prolonged hot tub. They are to be warned to take cooler baths and preferably to have someone within easy call. The bathroom door must not be locked.

When the skin is abnormally dry especially in elderly persons in the winter time excessive bathing may initiate or aggravate *pruritus*.

Partial Baths—Partial bathing is employed for a portion of the body *i.e.*, the *sitz baths* in rectovaginal conditions or prostatic inflammation and the *arm and foot baths* for local disturbances. The temperatures of these baths may be more extreme and medicaments can be used in greater concentration.

B Wet Dressings—The wet dressing produces a variety of effective actions. These include *cleansing*, *protection*, *drainage*, *irritation* or *counterirritation*, *maceration*, *local anesthetic* or *antipruritic effects* and *astringent*, *antiseptic*, *bactericidal*, *disinfectant* or *deodorant actions*. Wet dressings are applied *cold* or *hot*, *open* or *unprotected* or *closed* and *protected*. Hematomas, contusions and acute inflammatory conditions with exudation are usually made more comfortable by the *cold wet dressing*. Other wise most patients prefer the *hot or warm wet dressing*.

The wet dressing is applied on cotton, gauze, flannel or toweling. A large bulky but not heavy dressing is preferable. The material is wrung out so that it is "dryish". The temperature of the dressing is maintained by the local use of an ice bag, ice collar or a hot water bag. *Unless an electric pad is specifically and especially insulated it should not be employed since a short circuit and an electric burn may result.*

Open Dressings—The open or unprotected wet dressing is changed frequently or the solution is continuously replenished. The open wet dressing produces little maceration.

Closed Dressings—The closed wet dressing is covered by rubber tissue, oiled silk or cellophane to prevent evaporation. The intact skin is protected with an ointment or oily substance to prevent excessive maceration. If the closed wet dressing is to be bandaged the gauze is laid on loosely lest when it becomes wet through the shrinkage produce constriction and discomfort.

Protective and Cleansing Wet Dressings—The simplest of the wet dressings are *fresh milk*, *physiologic saline solution* and *saturated boric acid solutions*. These function as protectives and cleansing agents.

Astringent, Keratolytic and Anidrotic Wet Dressings—A mildly astringent dressing is prepared with (1) solution of aluminum subacetate diluted 1:15 to 1:30 with water or (2) solution of aluminum acetate (Burrow) diluted 1:10 to 1:20 with water. These solutions are *keratolytic* and *anidrotic*.

Antiseptic and Bactericidal Wet Dressings—The most effective antiseptic and bactericidal wet dressings are solutions of penicillin (1 cc = 250 units) and sulfanilamide (0.8 per cent) or sulfathiazole (5 per cent) in sterile distilled water or saline. These preparations have replaced saturated solution of boric acid, 1:1000; iodide of mercury (1:2000); silver nitrate (1:4 to 1 per cent); sulfured lime (*Vlemmckx's*) diluted 1:10 or 1:30 and *Epsom salts* (25 per cent).

Analgesic Wet Dressings.—Five per cent *magnesium sulfate* (Epsom salt) is astringent, mildly analgesic and antipruritic.

Deodorant Wet Dressings.—*Potassium permanganate* 1:4000 to 1:10000 is a mild bactericide and deodorant. *Hydrogen peroxide* in 25 per cent strength is used on foul smelling anaerobic wounds.

Antipruritic Wet Dressings.—Local itching is allayed by wet dressings of bicarbonate of soda, milk saline solution or boric acid.

C. Washes.—When the involved area cannot be bandaged, solutions or lotions are dabbed or painted on the skin and permitted to evaporate. The addition of glycerin to the solution adds body and prevents excessively rapid evaporation. The *shake lotion* is discussed elsewhere (p. 3147) since it is essentially a method of coating the skin with an insoluble powder.

Aqueous and Hydro-alcoholic Washes.—Solutions or washes are commonly prepared with plain water, rose water, lime water or alcohol. A volatile oil such as lavender, rosemary, bergamot, menthol or camphor gives color and/or odor to the preparation.

Astringent solutions are prepared with boric acid (5 per cent), tannic acid (1 per cent) or zinc sulfate (1 per cent). *Hydroalcoholic solutions* are mostly rubefacient. The addition of menthol in 0.25 to 1 per cent concentration will increase the cooling and refrigerant effect.

As an antipruritic, the concentration of menthol is increased to 2 per cent or phenol (0.25 to 1 per cent) is added.

| | |
|-----------------------|-------------|
| B. Menthol | 0.5 |
| Phenol | 5.0 |
| Salicylic Acid | 4.0 |
| Glycerin | 40.0 |
| Alcohol (80 per cent) | qs ad 240.0 |

Alkaline solutions prepared with 3 per cent bicarbonate of soda or lime water are somewhat sedative and antipruritic. A *keratolytic solution* is prepared with 3 per cent salicylic acid in alcohol. *Potassium permanganate* in 1:4000 to 1:1000 solution or a saturated solution of aluminum acetate containing a volatile oil for perfumery is employed for deodorant and anidrotic effects. *Stomatizing and keratoplastic solutions* are prepared with 3 per cent of liquid tar (Liquor Carbonis Detergens N.F.). Most *hair tonics* (p. 3141) are made by the use of cantharidin in 5 or 10 per cent hydro-alcoholic solution together with glycerin and a volatile oil. Boric acid is a mild antiseptic wash while bichloride of mercury 1:2000 is powerfully bactericidal.

Tinctures.—The tinctures are relatively concentrated solutions of active ingredients in alcohol. They should only be applied to a limited area of the skin. The use of excessive amounts of a tincture may produce (1) constitutional disturbances from the absorption of the active ingredients, (2) a local dermatitis as the result of idiosyncrasy or the primarily irritating action of the active agent.

Most commonly used is the tincture of *sodium* which is a 7 per cent solution. *Tincture of Larkspur* is employed for the relief of pediculosis of the scalp or pubic regions.

THE FAT SOLUBLE PREPARATIONS FOR LOCAL APPLICATION TO TEGUMENTARY STRUCTURES

The fat soluble preparations include the emulsions or liniments and the various ointments, salves, creams and oils.

D. Emulsions and Liniments.—Emulsions or liniments contain (1) oily or fatty substances suspended in aqueous or other liquid vehicles, (2) an aqueous solution that has been suspended in an oily medium.

For the most part, the emulsion or liniment is used on an acutely inflamed surface such as a burn, a dermatitis or a weeping eczema with vesiculation. It is best applied to large areas with a soft varnish brush.

The simplest and one of the best of the emulsions is milk.

Healing Baths—Sulfur baths are popularly used at many of the spas. They are employed for their alleged deep effect on the skeletal system and for their efficacy in generalized dermatoses. Sulfur baths are accomplished in the home by adding 50 to 150 gm. of crude potassium sulfate to the tub. This is a foul smelling mixture which stains the tub. It is not to be suggested for routine use in the home since the efficacy of the measure is questionable to say the least. Baths with *Vlemmckx's solution* (solution of sulfured lime) 150 cc. to the tub are valuable in treating widespread pyogenic infections particularly *pustular folliculitis* in hirsute individuals.

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B. Wet Dressings—The wet dressing produces a variety of effective actions. These include *cleansing protection drainage irritation or counterirritation maceration local anesthetic or antipruritic effects* and *astringent bactericidal disinfectant or deodorant actions*. Wet dressings are applied *cold or hot open or unprotected or closed and protected*. Hematomas contusions and acute inflammatory conditions with exudation are usually made more comfortable by the *cold wet dressing*. Other wise most patients prefer the *hot or warm wet dressing*.

The wet dressing is applied on cotton gauze flannel or toweling. A large bulky but not heavy dressing is preferable. The material is wrung out so that it is *dryish*. The temperature of the dressing is maintained by the local use of an ice bag ice collar or a hot water bag. *Unless an electric pad is specifically and especially insulated it should not be employed since a short circuit and an electric burn may result.*

Open Dressings—The open or unprotected wet dressing is changed frequently or the solution is continuously replenished. The open wet dressing produces little maceration.

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Antiseptic and Bactericidal Wet Dressings—The most effective antiseptic and bactericidal wet dressings are solutions of penicillin (1 cc. = 250 units) and *salicylanilide* (0.8 per cent) or *sulfathazole* (5 per cent) in sterile distilled water or saline. These preparations have replaced saturated solution of boric acid, *bellonine* of mercury (1:2000), silver nitrate (1½ to 1 per cent), sulfured lime (*Vlemmckx's*) diluted 1:10 or 1:50 and Epsom salts (25 per cent).

Analgesic Wet Dressings—Five per cent *magnesium sulfate* (Epsom salt) is astringent, mildly analgesic and antipruritic

Deodorant Wet Dressings—*Potassium permanganate* 1:4000 to 1:10000 is a mild bactericide and deodorant. *Hydrogen peroxide* in 2.5 per cent strength is used on foul smelling anaerobic wounds

Antipruritic Wet Dressings—Local itching is allayed by wet dressings of *bicarbonate of soda*, *milk saline solution* or *boric acid*

C. Washes—When the involved area cannot be bandaged, solutions or lotions are dabbed or painted on the skin and permitted to evaporate. The addition of glycerin to the solution adds body and prevents excessively rapid evaporation. The *shake lot ion* is discussed elsewhere (p. 3147) since it is essentially a method of coating the skin with an insoluble powder

Aqueous and Hydro-alcoholic Washes—Solutions or washes are commonly prepared with plain water, rose water, lime water or alcohol. A volatile oil such as lavender, rosemary, bergamot, menthol or camphor gives color and/or odor to the preparation.

Astringent solutions are prepared with *boric acid* (5 per cent), *tannic acid* (1 per cent) or *zinc sulfate* (1 per cent). *Hydroalcoholic solutions* are mostly rubefacient. The addition of menthol in 0.25 to 1 per cent concentration will increase the cooling and refrigerant effect.

As an antipruritic the concentration of menthol is increased to 2 per cent or phenol (0.25 to 1 per cent) is added.

| | |
|-----------------------|-----------|
| B. Menthol | 0.5 |
| Phenol | 50 |
| Salicylic Acid | 40 |
| Glycerin | 400 |
| Alcohol (60 per cent) | qs ad 400 |

Alkaline solutions prepared with 5 per cent bicarbonate of soda or lime water are somewhat sedative and antipruritic. A *keratolytic solution* is prepared with 5 per cent salicylic acid in alcohol. *Potassium permanganate* in 1:4000 to 1:1000 solution or a saturated solution of *aluminum acetate* containing a volatile oil for perfumery is employed for deodorant and *antidrotic effects*. *Stimulating and keratoplastic solutions* are prepared with 5 per cent of liquid tar (*Liquor Carbonis Fungicidus* N.F.). Most *hair tonics* (p. 3141) are made by the use of *cantharides* in 5 or 10 per cent hydro-alcoholic solution together with glycerin and a volatile oil. *Boric acid* is a mild antiseptic wash while *bichloride of mercury* 1:2000 is powerfully *bactericidal*.

Tinctures—The tinctures are relatively concentrated solutions of active ingredients in alcohol. They should only be applied to a limited area of the skin. The use of excessive amounts of a tincture may produce (1) constitutional disturbances from the absorption of the active ingredients, (2) a local dermatitis as the result of vasoconstriction or the primarily irritating action of the active agent.

Most commonly used is the tincture of *iodine* which is a 7 per cent solution. *Tincture of Larkspur* is employed for the relief of *pediculosis* of the scalp or pubic regions.

THE FAT SOLUBLE PREPARATIONS FOR LOCAL APPLICATION TO TFGUMENTARY STRUCTURES

The fat soluble preparations include the emulsions or liniments and the various ointments, salves, creams and oils.

D. Emulsions and Liniments—Emulsions or liniments contain (1) oily or fatty substances suspended in aqueous or other liquid vehicles, (2) an aqueous solution that has been suspended in an oily medium.

For the most part, the emulsion or liniment is used on an acutely inflamed surface such as a burn, a dermatitis or a weeping eczema with vesiculation. It is best applied to large areas with a soft varnish brush.

The simplest and one of the best of the emulsions is *milk*.

ointments are also used in the treatment of *seborrhea of the scalp* with dandruff for example

| | | |
|---|---------------------|-----------|
| R | Precipitated Sulfur | 30 |
| | Salicylic Acid | 18 |
| | Resorcin | 18 |
| | Castor Oil | 60 |
| | Petrolatum | qs ad 600 |

or

| | | |
|---|---------------------|-----------|
| R | Precipitated Sulfur | 30 |
| | Salicylic Acid | 18 |
| | Oil of Cade | 20 |
| | Benzoinated Lard | qs ad 600 |

Other Ointments—A 12 per cent ointment of *Balsam of Peru* is an effective parasiticide for use in scabies. *Ammoniated mercury ointment* (10 per cent) is both antiseptic and bactericidal. Thirty per cent calomel ointment is spirocheticidal (venereal prophylaxis). For keratoplasia a 3 to 10 per cent tar preparation may be used in an ointment base.

INSOLUBLE PREPARATIONS FOR LOCAL OR TOICAL APPLICATION TO TEGUMENTARY STRUCTURES

Insoluble preparations are applied to the skin as the pure chemical as a powder a poultice a plaster a paste or the shake lotion which on evaporation of the solvent leaves a film of the insoluble material

F Powders—Powders are used liberally over the entire skin surface. They serve their greatest purpose in the folds of the skin. Here they are *protective* and *absorbent*. The plain unscented *French chalk* is the least expensive and makes an excellent powder. The more expensive talcums contain both scent and coloring matter from which patients occasionally develop a *contact dermatitis*.

In applying powder to a fold in the skin the excess is removed with a dab of cotton test cake and local irritation occurs. The community powder puff is distinctly unhygienic. Obese patients with apron panniculus pendulous breasts and rubbing thighs men with large scrotums and children who perspire freely should powder well as part of the hygiene of the bath. Careful interdigital drying and a sprinkle of fungicidal powder prevent most *dermatophytoses*.

Ordinary talcum and zinc oxide powder are absorbent and anidrotic. Powdered boric acid (10 per cent) is mildly disinfectant. Powdered tannic acid is astringent. The addition to the powder of 0.25 to 1 per cent menthol or camphor is cooling and antipruritic. DDT powder is an effective pediculicide.

Powdered sulfur is used as an insecticide. Crystals of sulfanilamide and sulfathiazole exert a powerful bacteriostatic effect. Salicylic acid powder in 1 to 5 per cent concentration is an idrotic antipruritic and fungicidal. Aristol powder is bactericidal as is bismuth tribromphenat (Xeroform). Perfumed talcs are deodorant.

G Shake Lotions—Lotions are liquid preparations for use on the skin and exposed surfaces. They contain ingredients in solution suspension or both.

The shake lotion is widely used in dermatologic treatment as a convenient form of application of an insoluble powder (Unna Calamine White Lotions). The basic shake lotion is protective. The Unna and Calamine lotions are prepared as follows:

| Unna | | Calamine | |
|------|------------|----------|-------------------|
| R | Zinc Oxide | R | Prepared Calamine |
| | Talcum | | Zinc Oxide |
| | Glycerin | | Glycerin |
| | Water | | Lime Water |
| | aa 200 | | 100 |
| | 150 | | 100 |
| | qs ad 1200 | | 20 |
| | | | qs ad 1200 |

The addition of 0.5 to 1 per cent menthol makes either lotion mildly antipruritic and 0.5 to 2 per cent liquefied phenol more strongly so.

E Ointments Creams Salves and Oils—Ointments salves creams and oils are widely employed in dermatology. They are prepared with a base of animal or vegetable oil mineral grease or mineral oil. The animal and vegetable oils include ordinary *lard sheep wool fat (lanolin) goose grease and mutton fat castor cottonseed and olive oil*. These are apt to become rancid and in this respect alone are inferior to the mineral preparations.

Ointment Bases—Ointment bases are of several varieties (Sulzberger). They are arranged in order of penetration diminishing from greatest to least power.

1 *Cholesterol or Lipoid Creams* (Aquaphor Almatone). The addition of cholesterol increases penetrating power.

2 *Hydrous Wool Fat (Lanolin)* which can absorb twice its weight of water.

3 *Benzoinated Lard* in which the benzoin lessens the tendency to rancidity.

4 *Petrolatum* (yellow or white).

5 *Boric Acid Ointment* (3-10 per cent) in petrolatum.

6 *Cold Cream* (Ung. Aquae Rosae).

7 *Zinc Oxide Ointment* (25 per cent) in petrolatum.

8 *Zinc Oil* zinc oxide and mineral or olive oil in equal parts.

9 *Vanishing Cream* (Warren).

| | |
|---------------------|------|
| Stearic Acid | 12.5 |
| Glycerin | 6.5 |
| Distilled Water | 75.0 |
| Potassium Carbonate | 1.2 |

10 *Soft Soap or Tincture of Green Soap*

The main purpose of the fat or oily base is its *greater penetrating power* through the various layers of the skin. To promote penetration the preparation should be *rubbed vigorously* into the involved area. The oily bases penetrate the skin and hold the medication in intimate and prolonged contact with the lesion. Simultaneously they soften and lubricate the skin. For these functions they are vastly superior to preparations dissolved in water and/or alcohol.

Ointments should not be used on oozing or infected areas. They form a coating that may prevent drainage and favor anaerobic conditions conducive to the growth of harmful bacteria such as the tetanus organism. Since they retain heat by insulation they tend to *increase itching and burning*.

The ointment is held in place by means of a bandage. The ointment is not to be placed directly on the bandage but may be spread over the skin with a tongue blade or buttered onto a gauze linen or flannel pad. Ointment should be removed at least once daily with cotton soaked with mineral or olive oil and replaced if necessary.

The following prescriptions suggest the various uses for the oil soluble preparations.

Protective Ointments—Boric Acid (10 per cent) or Zinc Oxide (20 per cent)

Antipruritic Ointments

0.5 per cent Menthol in Protective Ointment

0.25 to 1 per cent Phenol in Protective Ointment

5 to 10 per cent Salicylic Acid

5 to 10 per cent Benzocaine (local anesthetic)

| | |
|---------------------|--------------|
| R. Menthol | 0.1 |
| Phenol | 0.6 |
| Tar Oil | 2.0 |
| Benzocaine | 4.0 |
| Zinc Oxide Ointment | q.s. ad 60.0 |

Keratolytic Ointment—A keratolytic ointment is the popular "Whitfield's Ointment" which contains 6 per cent salicylic acid and 12 per cent benzoic acid in the ointment base. This is also an effective ointment when employed as a *fungicide*. Keratolysis is also accomplished with resorcin 1 to 5 per cent and/or precipitated sulfur (1 to 10 per cent) in zinc oxide ointment or cold cream base. These keratolytic

1 **Face and Neck Preparations**—Face and neck preparations include the bulk of cosmetics since these areas are most exposed to the critical eye

1 *Creams and pastes* are purveyed in almost infinite variety with claims frequently extravagant and illogical Basically they consist of varied combinations of animal oil (lanolin beeswax) vegetable oil (castor olive) mineral oil water and perfume

The proper addition of stearic acid triethanolamine and potassium hydroxide alters the basic cream into a *vanishing cream*

The addition of lecithin and/or cholesterol enhances absorptive powers and converts the cream into one of the *lubricating type* There is no especial virtue in this cream beyond its action as an emollient on dry and scaling skin

Astringent creams are produced by the addition of alum zinc sulfate or zinc oxide

Bleaching creams (freckle removers) contain as active agents hydrogen peroxide lactic or citric acid or the peroxides of zinc or magnesium Preparations containing ammoniated mercury are dangerous since repeated application may lead to a permanent dirty gray pigmentation of the skin due to deposition of metallic mercury

2 *Face powders* consist of a covering agent (zinc oxide titanium dioxide) a slip material (talc) an adhesive (zinc or magnesium stearate or starch) an absorbent (precipitated chalk magnesium carbonate or purified kaolin) a coloring material (ochre burnt senna brilliant pink like) and a perfume oil By varying the proportions heavy or light powders of different color shades are produced

The liquid cream and cake powders are essentially the same except that they are produced in the physical form indicated The powders are applied to the face neck and arms to give a homogenous appearance in evening dress The *liquid* form is suspended in water and glycerin The *cream* form is made with a vanishing cream base

3 *Rouges* are made in the form of creams pastes and compacts The *creams* consist of a cold cream type base in which is incorporated the desired cosmetic lake color and a perfume More satisfactory are the *paste rouges* which have a more solid base similar to a lipstick *Compact rouge* consists of a base similar to face powder containing the cosmetic lake color bound together with tragacanth or karaya gum A preservative must be added

4 *Bleaches* may be made as creams (see 1 above) lotions or powders Lotions are prepared with hydrogen peroxide tincture of benzoin rose water perfume and a minute amount of preservative They may also consist of lactic acetic or citric acids in hydroalcoholic solution Concentrated lemon juice will remove freckles A powder form is zinc peroxide sodium perborate magnesium carbonate and starch

5 *Astringent creams* have already been described (No 1 above) Lotions usually contain alum zinc sulfate and glycerin in a scented hydroalcoholic solution Their effect is transient and illusory

Skin toning lotions are weak astringents similar to the astringent lotions already described They are of equally dubious value

6 *Wrinkle removers* obviously are unable to perform the task for which they are recommended Some consist of exotic agents like *turtle oil* blended with other animal and vegetable oils Others are cold creams enriched with cholesterol and lecithin Recently *estrogenic ointments* have been widely promoted for restoring youthful skin

7 *Facial masks* have a tightening effect upon the skin They are supposed to cleanse the pores of impurities and remove the superficial dead layers of the skin It would seem that thorough cleaning with soap and water more simply and cheaply accomplishes the same aims The masks are made from a base usually combined with other less active ingredients as dextrine and cetyl alcohol in a hydroalcoholic base containing perfume preservative and menthol The *clay packs* consist of purified kaolin kieselguhr (Purified Siliceous Earth USP) calcium mucilage glycerin alcohol water and perfume

8 *Depilatories* are used by women to remove hair from the face armpits and legs The salts most commonly employed are the sulfides of barium calcium potassium and sodium These are frequently productive of considerable irritation and in sensitive individuals may evoke marked *dermatitis*

Three to 10 per cent benzocaine functions as a more efficient antipruritic and local anesthetic Stimulation of the skin and keratoplasmia may be obtained with 3 to 40 per cent Liquor Carbonis Detergens

| | |
|------------------------|------------|
| I; Menthol | 0.3 |
| Phenol | 1.2 |
| Liq Carbonis Detergens | 100 |
| Benzocaine | 60 |
| Lotio Calamine | qs ad 1200 |

Keratolysis results from the addition of 1 to 3 per cent *salicylic acid* *resorcinol* (2 to 10 per cent) or *precipitated sulfur* 3 to 10 per cent *Cinnabar* 2 to 5 per cent may be added for bactericidal action

The shake lotion is best applied to the skin with a soft clean flat paintbrush When the lotion dries the insoluble substance remains in place The shake lotion has all the functions and uses of powders and the advantage of greater adhesiveness

- H **Pastes**—Pastes are semisolid preparations consisting of insoluble highly dispersed powders in a grease base The paste does not tend to absorb secretion and is more permeable It may be used at times on *acutely inflamed areas of skin* that show *crusting* or *oozing* Active ingredients in a paste are less potent than in an ointment *ie* 3 per cent *salicylic acid* in a paste will produce less effect than the same amount in an ointment

An example of a simple paste is *zinc oxide* and *talcum* or *cornstarch* finely powdered 15 parts of each and *petrolatum* 30 parts (*Lassar's Paste*) This is purely protective in function Active ingredients such as *salicylic acid* tar and sulfur may be added to this paste

- I **Plasters**—Plasters are masses which adhere to the skin They may be simple or medicated The popular *mustard plaster* and the *corn plaster* are obvious examples The first is rubefacient and counterirritative the latter *keratolytic*

- J **The Direct Application of Chemicals**—Chemicals may be applied directly to the skin *Silver nitrate* pencil burns *exuberant granulation* *Chromium trioxide* and *nitric acid* destroy moles or warts Pure *phenol* may be used on the unbroken skin to open small *boils* or *furuncles* A slush of *carbon dioxide* is an effective *keratolytic* *Trichloroacetic acid* may be used to remove *warts* and *xanthelasma*s of the eyelids but must be applied with caution

COSMETICS

While cosmetics are rarely ordered by the physician knowledge of them is important because of the frequency with which they may produce *abnormal cutaneous reactions* The most common condition they induce is *dermatitis venenata* so called *contact dermatitis* (p 3330)

For the general information of the practitioner the widely and commonly employed cosmetic preparations are described They are grouped as far as possible according to the location where they are applied

- I Face and neck preparations
- II Eye preparations
- III Hair and scalp preparations
- IV Lip and mouth preparations
- V Hand preparations
- VI Nail preparations
- VII Armpit preparations
- VIII Foot preparations
- IX Body preparations
- X Miscellaneous preparations

3 *Eyebrow pencil* is a hard crayon composed of carbon black, in a base of paraffin

4 *Eyelash and eyebrow pomade* is a simple emollient of peach kernel oil, paraffin petrolatum perfume and a modicum of preservative

5 *Eye washes* for cosmetic purposes are claimed to relieve eyestrain and make the eyes more lustrous. They are essentially weak alkaline scented boric acid solutions. Occasionally a mild astringent like zinc sulfate is added

III Hair and Scalp Preparations—1 *Shampoos* are of various types (soap shampoos soapless and dry)

Soap shampoos lather freely cleanse thoroughly and contain only small amounts of free alkali. These soaps are liquids and are made of vegetable oils (cocoanut olive palm kernel sesame) saponified with potassium and sodium hydroxide and perfumed

Soapless shampoos are neutral or slightly acid. Generally they consist of sulfonated castor and olive oils. They are excellent cleansers and do not produce the marked dryness of the alkaline shampoos. Commercial products include Tersus (Doak Co.) Acidolate Drene and Admiracion

Dry shampoos are (1) soapless (talc magnesum carbonate and wheat starch) or (2) soapy (powdered potash soap sodium sesquicarbonate potassium carbonate and borax)

2 *Hair tonics* sold for the cure of alopecia or to arrest the falling of the hair are of varied composition. Their common ground is inability to perform the function which is claimed for them. They contain one or more of the following drugs: resorcinol betanaphthol chloral hydrate quinine pilocarpine hydrochloride castor oil tincture of cantharides spirits of formic acid. The base is usually scented alcohol

Preparations marketed for the treatment of *dry scaly scalps* usually consist of a cleanser as sulfonated castor oil an irritant as cantharides or formic acid and an antiseptic as chlorothymol in hydroalcoholic solution. For *oily scaly scalps* products containing quinine bay rum resorcinol and so on are sold

3 *Curling and Waving Materials*—The former consist basically of *gummy substances* in water. Substances most widely used are quince seed and karaya gum. *Permanent waving* may be done with a solution of ammonia, borax and water. More complex formulas are also used in this procedure

4 *Brilliantines* are used to make the hair glossy and to maintain it in position. Most are liquid perfumed preparations of mineral oil or vegetable oils. Some are jellies of mineral oil spermaceti white paraffin stearic acid and perfume

5 *Hair straighteners* may be waxy (of beef tallow white beeswax odorless castor oil and perfume) or *gummy* in which mucilage of tragacanth plays the important role

6 *Hair bleaches* are used to lighten the color of normal scalp hair or of excessive growths of hair on the face arms and legs. Combinations of hydrogen peroxide and strong ammonia are most frequently used. Lemon juice will perform the same function

7 *Bluing of white hair* is done with a solution of 0.15 per cent aniline violet in water

8 *Dyeing or restoring of the hair* may be done with several different types of preparations. Least harmful are the so-called henna rinses which consist of ground henna leaves in a perfumed hydroalcoholic vehicle. Dangerous are the preparations of *silver* and *lead* as they may lead to metallic poisoning

The most satisfactory medium is the *aniline dye* either paraphenylenediamine or one of its derivatives. However there is an element of risk in this because of its rather high index of sensitization and production of contact dermatitis. Preliminary patch tests should always be done to eliminate so far as possible the presence of idiosyncrasy. It should never be applied to a scalp where open wounds are present since absorption may lead to serious intoxication even agranulocytosis or liver damage

IV Lip and Mouth Preparation—*Lip pomades* consist of white beeswax benzoated lard same and castor oil and perfume oil. They may be much more complex than this. The lip pomades are used for dry chapped lips. *Lipsticks* are basically lip

Thallium acetate acts as a depilatory hematogenously. Its action is limited to the scalp and eyebrows. It is of no value as a cosmetic preparation. Its use must be frowned upon generally since it is a potent poison capable of serious intoxication even death. It is used by some dermatologists to epilate the scalp in cases of *tinea capitis* in children.

The *epilating waxes* are perfumed combinations of rosin, beeswax, paraffin and petrolatum. These are not actually depilatories but act as a mechanical device for removing numbers of hairs en masse. *Pumice stones* are used to rub away excessive hairs on the face.

There is no doubt that the only safe effective method of permanent epilation is by *electrolysis* with galvanic or coagulating current (p. 3,02). The use of the *roentgen ray* for permanent epilation is dangerous frequently producing serious *radiodermatitis*. No reputable dermatologist uses this last procedure.

The *chemical depilatories* are most popular as pastes and powders. The *paste* contains 15 per cent each of barium and strontium sulfide, talc, cornstarch, glycerin, water and perfume. The *powder* 35 per cent barium sulfide with titanium dioxide, wheat starch, menthol and perfume.

9 *Sun preparations* are sold for three distinct purposes: (1) to prevent sunburn, (2) to simulate sun tanning, and (3) to treat sunburn.

Sunburn protectives are important medically because of their value in light sensitive persons, particularly patients with *lupus erythematosus*. Many substances have some screening effects against the ultraviolet rays of the sun, notably menthyl salicylate, quinine salts and salol. Most vegetable oils, waxy ointments and calamine lotion afford some degree of protection.

Protectives may be prepared as creams:

| | |
|--------------------|------|
| R. Quinine Sulfate | 5.5 |
| White Ceresin Wax | 5.5 |
| White Petrolatum | 90.5 |
| White Mineral Oil | 19.5 |
| Lanolin | 15.0 |
| Water | 35.5 |
| Oil of Cassia | 0.5 |

or

| | |
|-----------------------|------|
| R. Menthyl Salicylate | 14.0 |
| Borax | 1.8 |
| Beeswax | 20.0 |
| White Mineral Oil | 20.0 |
| Water | 28.7 |
| Petrolatum | 15.0 |
| Perfume | 0.5 |

The lotion is simpler: as 11.5 per cent menthyl salicylate, alcohol 10 per cent, glycerin 5.0 per cent, water 73.25 per cent and perfume 0.25 per cent. Quinine bisulfate may be incorporated into a suntan face powder.

Preparations to simulate sunburn are chiefly suntan face powders. These are ordinary face powders with a disproportionately large content of golden ochre. Creams and oils are also made using a pigment brown color to achieve the desired effect.

Remedial preparations for sunburn are soothing ointments containing mild antiseptics and local anesthetics. Some of the chemicals incorporated, capable of inducing untoward reaction, are ethyl aminobenzoate, oxyquinoline sulfate, picric acid, linseed oil, butesin and nupercaine.

- II *Eye Preparations*—1 *Eye shadow* is usually made in three shades: brown, blue and green. The base consists of white petrolatum, cetyl alcohol, lanolin, white beeswax and spermaceti. Cosmetic blue lake produces the blue shade, burnt amber the brown, cosmetic green lake the green.

2 *Mascara* is used most widely in the cake form and applied with a moist brush. It consists of prepared lamp black combined with glyceryl monostearate, triethanolamine, stearic acid, petrolatum, gelatin, water and beeswax.

These ingredients may also be produced as pastes powders compacts and sticks The pastes are easily applied and may be simply an aluminum salt in a vanishing cream base

2 *Depilatories*—see under I Face and Neck part 8 In the axillae and on the legs the most widely practiced procedure is shaving When done carefully and cleanly this produces very satisfactory results

VIII *Foot Preparations*.—1 *Deodorants and astringents* are used rather widely on the feet, practically always in powder form They may be largely talc containing 15 to 30 per cent boric acid an active ingredient may be added such as 1 to 3 per cent salicylic acid 3 to 5 per cent tannic acid zinc peroxide 10 per cent or 50 per cent aluminum acetate

An astringent deodorant may be prepared

| | |
|----------------|------|
| R Water | 54.0 |
| Alcohol | 33.0 |
| Glycerin | 10.0 |
| Formalin | 0.5 |
| Chlorothymol | 0.2 |
| Salicylic Acid | 2.0 |
| Perfume Oil | 0.25 |

These powders also have protective powers against fungus infection of the feet and are valuable in daytime treatment of such infections

2 *Commercial corn removers* are collodons or plasters heavily impregnated with salicylic acid

IX. *Body Preparations*.—1 Powders for body use are generally somewhat heavier than face powders and are uncolored A typical powder is talc 50.0 kaolin 30.0 boric acid 2.0 precipitated chalk 17.0 and perfume 1.0

2 *Massage* may be performed with powder alcohol oil of theobroma and similar agents *Greaseless massage* preparations are marketed and are composed of vanishing cream glycerin cornstarch water and witch hazel These have no particular virtue since the value of massage lies in the mechanical procedure involved rather than the lubricant employed

X *Miscellaneous Preparations*.—1 *Perfumes and toilet waters* are complex products consisting of hydroalcoholic solutions of essences obtained from flowers and fixatives of animal origin Most perfumes today are prepared from synthetic scents

2 *Shaving preparations* include creams soaps beard softeners after shaving lotions and powders *Shaving cream* is a nearly neutral mixture of potassium and sodium stearate to produce a rich lather and soften the hairs *Cocoonut oil* increases the richness of the lather *glycerin* prevents rapid dehydration and acts as a lubricant Wetting action is increased by adding *triethanolamine* Perfumed water forms about 45 per cent of the product

Bruless shaving creams are modified vanishing creams Principle ingredients are the fatty acids (stearic) alkali (potassium hydroxide) and water *Cetyl alcohol* and *triethanolamine* are added to reduce surface tension *glycerin* and *mineral oil* to allay irritation

Beard softeners are not widely used They are applied before shaving before applying the shaving cream An example is sodium cholate 0.35 per cent sodium lauryl sulfate 2.35 per cent glycerin 7.0 per cent water 89.8 per cent and perfume 0.1 per cent

After shaving lotions are mildly styptic and antiseptic as

| | |
|--------------|------|
| R Alcohol | 90.0 |
| Zinc Sulfate | 0.5 |
| Glycerin | 5.0 |
| Benzocaine | 0.2 |
| Perfume | 0.3 |
| Water | 4.0 |

pomades to which have been added such dyes as cosmetic scarlet lake brilliant red lake maroon lake. Some dyes as fluorescein and eosin have photosensitizing properties.

- V **Hand Preparations**—1 *Creams* of various sorts are sold especially for use on the hands. These are usually cold creams vanishing or lubricating creams (p 3139). They are recommended to keep the hands soft and prevent chapping. They usually include some softening materials as cetyl alcohol, cocoa butter or gums.

2 *Lotions* are used for the same purposes as creams. They are marketed under such names as honey and almond, cucumber and strawberry lotions and consist chiefly of gums of quince seed or karaya with glycerin, soap, alcohol and water and a preservative. An inexpensive and effective hand lotion has been described (p 3131).

3 *Liquid hand soaps* are exemplified by the following: cottonseed oil, sodium and potassium hydroxide, coconut oil, alcohol, perfume and distilled water.

- VI **Nail Preparations** 1 *Nail polish* is sold in various forms, the most popular being the nail lacquer or enamel. Enamels are composed of nitrocellulose, ethyl or butyl acetate, diethyl phthalate, camphor and a dye.

Nail polish powder may be tin oxide, titanium dioxide and oleic acid. The nail polish paste may consist of tin oxide, carnauba wax, white beeswax, petrolatum and titanium dioxide.

2 *Nail polish remover* is a solvent as a mixture of ethyl and butyl stearate or one of castor oil, dibutyl phthalate and ethyl acetate.

3 *Nail bleach (white)* is used under the free border of the nail. As a cream it may contain titanium dioxide, talc, zinc peroxide, petrolatum and mineral oil. As a liquid it may be oxalic acid, citric acid and rose water.

4 *Cuticle softener* is a complex of cholesterol, absorption base, petrolatum, beeswax, sulfonated castor oil, sodium lauryl sulfate, trisodium phosphate, water and perfume. *Cuticle remover* is essentially potassium hydroxide and glycerin in rose water.

- VII **Armpit Preparations**—1 *Deodorants and anidrotics* are grouped together since it is the decomposition of sweat by the skin bacteria which produces the unpleasant odor. There are of course instances in which the secreted sweat is inherently malodorous. This condition is termed *bromidrosis* (p 3462). Bathing will give temporary relief and perfumes and toilet waters to a certain extent conceal the odors. Most effective results follow the simple application of bicarbonate of soda.

The anidrotic and deodorant preparations contain an antiseptic, an anidrotic and a perfume. The most widely used anidrotics are aluminum chloride, alum and formalin.

Deodorant Solution

| | |
|---------------------|------|
| ℞ Aluminum Chloride | 15.0 |
| Water | 81.5 |
| Glycerin | 3.0 |
| Perfume | 0.5 |

or

| | |
|-------------------|------|
| ℞ Boric Acid | 4.0 |
| Formalin | 1.0 |
| Aluminum Chloride | 11.0 |
| Alcohol | 5.0 |
| Water | 78.5 |
| Perfume | 0.5 |

or

| | |
|-----------------------------|-------------|
| ℞ Aluminum Chloride | 26.0 |
| Ethyl Alcohol (95 per cent) | 60.0 |
| Acetic Ether | 0.7 |
| Oil of Clove | 0.07 |
| Oil of Rose | 0.5 |
| Dist. Water | qs ad 120.0 |

These ingredients may also be produced as pastes powders compacts and sticks. The pastes are easily applied and may be simply an aluminum salt in a vanishing cream base.

2 *Depilatories*—see under I Face and Neck part 8. In the axillae and on the legs the most widely practiced procedure is shaving. When done carefully and cleanly this produces very satisfactory results.

VIII Foot Preparations.—1 *Deodorants and anidrotics* are used rather widely on the feet, practically always in powder form. They may be largely talc containing 15 to 30 per cent boric acid. An active ingredient may be added such as 1 to 3 per cent salicylic acid 3 to 5 per cent tannic acid zinc peroxide 10 per cent or 50 per cent aluminum acetate.

An astringent deodorant may be prepared

| | |
|----------------|------|
| R Water | 54.0 |
| Alcohol | 33.0 |
| Glycerin | 10.0 |
| Formalin | 0.5 |
| Chlorothymol | 0.2 |
| Salicylic Acid | 2.0 |
| Perfume Oil | 0.25 |

These powders also have protective powers against fungus infection of the feet, and are valuable in daytime treatment of such infections.

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IX Body Preparations.—1 *Powders for body use* are generally somewhat heavier than face powders and are uncolored. A typical powder is talc 50.0 kaolin 30.0 boric acid 2.0 precipitated chalk 17.0 and perfume 1.0.

2 *Massage* may be performed with powder alcohol oil of theobroma and similar agents. *Greaseless massage preparations* are marketed and are composed of vanishing cream glycerin cornstarch water and witch hazel. These have no particular virtue since the value of massage lies in the mechanical procedure involved, rather than the lubricant employed.

X Miscellaneous Preparations.—1 *Perfume and toilet waters* are complex products consisting of hydro-alcoholic solutions of essences obtained from flowers and fixatives of animal origin. Most perfumes today are prepared from synthetic scents.

2 *Shaving preparations* include creams soaps beard softeners after shaving lotions and powders. *Shaving cream* is a nearly neutral mixture of potassium and sodium stearate to produce a rich lather and soften the hairs. *Cocoonut oil* increases the richness of the lather. *glycerin* prevents rapid dehydration and acts as a lubricant. Wetting action is increased by adding *triethanolamine*. Perfumed water forms about 45 per cent of the product.

Brushless shaving creams are modified vanishing creams. Principle ingredients are the *fatty acids* (stearic) *alkali* (potassium hydroxide) and water. *Cetyl alcohol* and *triethanolamine* are added to reduce surface tension. *glycerin* and *mineral oil* to allay irritation.

Beard softeners are not widely used. They are applied before shaving and before applying the shaving cream. An example is sodium cholate 0.35 per cent sodium lauryl sulfate 2.35 per cent glycerin 7.0 per cent water 89.8 per cent and perfume 0.5 per cent.

After shaving lotions are mildly styptic and antiseptic as

| | |
|--------------|------|
| R Alcohol | 20.0 |
| Zinc Sulfate | 0.5 |
| Glycerin | 5.0 |
| Benzocaine | 0.2 |
| Perfume | 0.3 |
| Water | 74.0 |

pomales to which have been added such dyes as cosmetic scarlet lake brilliant red lake maroon lake Some dyes as fluorescein and eosin have photosensitizing properties

- V **Hand Preparations**—1 *Creams* of various sorts are sold especially for use on the hands These are usually cold creams vanishing or lubricating creams (p 3137) They are recommended to keep the hands soft and prevent chapping They usually include some softening materials as cetyl alcohol cocoa butter or gums

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Nail polish powder may be tin oxide titanium dioxide and oleic acid The *polish paste* may consist of tin oxide carnauba wax white beeswax petrolatum titanium dioxide

2 *Nail polish remover* is a solvent as a mixture of ethyl and butyl alcohol or one of castor oil dibutyl phthalate and ethyl acetate

3 *Nail bleach (white)* is used under the free border of the nail As it may contain titanium dioxide talc zinc peroxide petrolatum and mineral oil As a liquid it may be oxalic acid citric acid and rose water

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- VII **Armpit Preparations**—1 *Deodorants and antiperspirants* are grouped together because of the decomposition of sweat by the skin bacteria which produces the odor There are of course instances in which the secreted sweat is itself odorless This condition is termed *bromidrosis* (p 3147) Bathing with soap and water relieves and perfumes and toilet waters to a certain extent conceal the odor The effective results follow the simple application of bicarbonate of soda

The antiperspirant and deodorant preparations contain an antiseptic and a perfume The most widely used antiperspirants are *aluminum chloride* *formalin*

Deodorant Solution

R Aluminum Chloride 1
Water
Glycerin
Perfume

or

R Boric Acid
Formalin
Aluminum Chloride
Alcohol
Water
Perfume

or

R Aluminum Chloride
Ethyl Alcohol (95 per cent)
Acetic Ether
Oil of Clove
Oil of Rose
Dist Water

These ingredients may also be produced as pastes powders compacts and sticks The pastes are easily applied and may be simply an aluminum salt in a vanishing cream base

2 *Depilator*—see under I Face and Neck part 8 In the axillae and on the legs the most widely practiced procedure is shaving When done carefully and cleanly this produces very satisfactory results

VIII *Foot Preparations*—1 *Deodorants and anidrotics* are used rather widely on the feet, practically always in powder form They may be largely talc containing 15 to 30 per cent boric acid an active ingredient may be added such as 1 to 3 per cent salicylic acid 3 to 5 per cent tannic acid zinc peroxide 10 per cent or 50 per cent aluminum acetate

An astringent deodorant may be prepared

| | |
|----------------|------|
| R Water | 51.0 |
| Alcohol | 33.0 |
| Glycerin | 10.0 |
| Formalin | 0.5 |
| Chlorothymol | 0.2 |
| Salicylic Acid | 2.0 |
| Perfume Oil | 0.25 |

These powders also have protective powers against fungus infection of the feet and are valuable in daytime treatment of such infections

2 *Commercial corn removers* are collodons or plasters heavily impregnated with salicylic acid

IX. *Body Preparations*—1 Powders for body use are generally somewhat heavier than face powders and are uncolored A typical powder is talc 50.0 kaolin 30.0 boric acid 2.0 precipitated chalk 17.0 and perfume 1.0

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Brushless shaving creams are modified vanishing creams Principle ingredients are the fatty acids (stearic) alkali (potassium hydroxide) and water *Cetyl alcohol* and triethanolamine are added to reduce surface tension glycerin and mineral oil to allay irritation

Beard softeners are not widely used They are applied before shaving i.e. before applying the shaving cream An example is sodium cholate 0.25 per cent, sodium lauryl sulfate 2.25 per cent glycerin 7.0 per cent water 89.8 per cent and perfume 0.5 per cent

4) *After-shave lotions* are mildly styptic and antiseptic as

| | |
|--------------|------|
| R Alcohol | |
| Zinc Sulfate | 20.0 |
| Glycerin | 0.5 |
| Benzocaine | 5.0 |
| Perfume | 0.2 |
| Water | 0.5 |
| | 74.0 |

After shaving powder is similar to face powder but somewhat heavier. It is also colored in different shades and scented.

3 *Bath preparations* include bath salts, powders, foam baths, milk baths and perfumed oil baths.

Bath salts are used presumably to soften water but probably largely because of the pleasant odor. Bath salts are 99 per cent sodium sesquicarbonate and 1 per cent perfume and color. Some of these are made to *effervesce* by adding sodium bicarbonate and tartaric acid. *Bath powders* are used on the body after the bath and are identical with body powders. *Foam powders* to produce a "bubble bath" are composed of sodium bicarbonate, tartaric acid, borax, lauryl sodium sulfate and perfume. *Simulated milk baths* are made by adding a mixture of anhydrous lanolin, glycerin, rose water, tincture of benzoin, mucilage of acacia, ethereal oil and perfume. *Pine baths* are made by adding a solution of 2 per cent boryl acetate in sulfonated olive oil. *Bath perfume* is 5 per cent eau de cologne compound in sulfonated olive oil.

4 *Antiseptic baby oil* consists of 0.2 per cent oxyquinoline sulfate and 0.1 per cent maleic anhydride in mineral and peanut oils.

5 Preparations to *conceal cosmetic blemishes* like angiomas, pigmentations and depigmentations are available commercially. They are prepared usually in liquid or cake form of different tints to match the normal skin. A popular brand is "Cover mark."

CHAPTER 143

THE TECUMENTARY SYSTEM: CONGENITAL SKIN DISEASES

| | |
|---|---|
| Acrodynia (Frythredema, Pink Disease) | Epidermolysis Bullosa |
| Adenoma Sebaceum | Fat Necrosis of the Newborn |
| Albinism (p 1560) | Ichthyosis |
| Angiomas Lymphangiomas Moles and Nevi (p 3200) | Keratosis Follicularis (Darier) |
| Auricular Fistulas | Keratoderma Palmaris et Plantaris |
| Congenital Alopecia and Hypertrichosis (p 3440) | Lymphedema of the Legs (Milroy's Disease) |
| Congenital Abnormalities of the Nails (p 3451) | Multiple Benign Cystic Epithelioma (p 3208) |
| Cutis Hyperelastica | Multiple Neurofibromatosis (von Recklinghausen's Disease) |
| Cutis Verticis Gyrate | Sclerema of the Newborn (Sclerema Neonatorum) |
| Dermatosis Papulosa Nigra | Syringocystoma (p 3209) |
| Fetodermal Defects | Urticaria Pigmentosa |
| Edeema of the Newborn (Edema Neonatorum) | Xeroderma Pigmentosum |

A **HETEROGENEOUS** group of skin conditions is of congenital origin. Included are *metabolic abnormalities atrophies and keratoses developmental defects pigmentations depigmentations and neoplasms*. Some of these affections are manifest at birth whereas others may not become apparent until later life. This group of disorders is not to be confused with the cutaneous abnormalities of the newborn (p 3146). The infant may not yet exhibit the lesions of a congenital disease but may suffer from a cutaneous manifestation of infection such as *prenatal syphilis* or from a metabolic disorder such as *vitaminosis K* (hemorrhagic disease of the newborn).

ACRODYNIA (ERYTHREDEMA PINK DISEASE)

Acrodynia is an uncommon disease most prevalent in the first two years of life. A *multiple vitamin deficiency* particularly of the B complex may be causative. It has been observed in association with the more serious abnormalities of the newborn such as *prenatal syphilis* (p 2787) *icterus neonatorum* (p 1067) and other severe systemic diseases.

Systemic Symptoms—Acrodynia is associated with a multiplicity of systemic symptoms which include *restlessness irritability malnutrition anorexia photophobia tachycardia and hypertension* suggesting a profound metabolic disorder.

Cutaneous Manifestations—The skin manifestations of pink disease consist of painful swollen red cold hands and feet. The nose may be red and the cheeks bluish red. Desquamation of the palms and soles may occur with secondary pyogenic infection. In addition there may be itching papular eruptions on the trunk profuse diaphoresis moderate alopecia and ulcerations of the buccal mucous membranes.

Course and Treatment—Acrodynia follows a prolonged course lasting several months as a rule. Death may occur from malnutrition or intercurrent disease. Prompt response is said to follow treatment with a high-

DIFFERENTIAL DIAGNOSIS OF

Common Dermatoses of Newborn and Infants

| DISTURBANCE | LESION | PREDILECTION | REMARKS |
|---|---|----------------------------|--|
| Acrodynia (p 3145) | Redness swelling | Hands and feet | Try response to vitamin B |
| Adenoma sebaceum (p 3148) | Papules telangiectases | Face | With mental deficiency and epilepsy |
| Albinism (p 1560) | Depigmentation | Generalized | Iris involvement |
| Angioma (p 3200) | Strawberry mark, Port wine stain | Nape of neck face and arms | Vascular anomaly |
| Atopic dermatitis (p 3342) | Infantile eczema | Face body and extremities | Seek offending allergen (milk, eggs) |
| Auricular fistula (p 3149) | Tract | In front of ear | |
| Contact dermatitis (p 3330) | Moist eczema | Especially diaper area | Distribution of eruption |
| Dermatitis medicamentosa (p 3335) | Maculo papules erythema | Generalized | History of administration of drug |
| Ectodermal defects (p 3150) | Atrophy | Face hair and nails | Saddle nose |
| Edema neonatorum (p 3151) | Swelling | Generalized or legs | With prematurity and marasmus |
| Epidermolysis bullosa (p 3151) | Bullae | At points of trauma | Produce by pinching |
| Exanthems | Scarlatiniform maculopapular vesiculopustular | Generalized | With fever and constitutional manifestations See tables pp 180 412 and 422 |
| Hemorrhagic disease of newborn (p 1111) | Echymoses purpuras | Skin and mucous membranes | Vitamin K deficiency |
| Hydroa aestivale (p 3176) | Vesicles and bullae | Exposed parts | After exposure to sun |
| Ichthyosis (p 3152) | Fish skin | Generalized or localized | |
| Icterus | Jaundice | Generalized | See table p 1955 |
| Impetigo contagiosa (p 3251) | Vesicles pustules and crusts | Exposed parts | Highly contagious staphylococcal invasion |
| Intertrigo (p 3161) | Acute inflammation | Diaper area | Identify contactual cause |
| Keratosis follicularis (p 3153) | Papules and greasy crusts | Generalized | Vitamin A deficiency |

| DISTURBANCE | LESION | PREDILECTION | REMARKS |
|------------------------------------|---|--|--|
| Moles and nevi (p 3204) | Tumors and papules | Anywhere | May be pigmented and/or hairy |
| Neurodermatitis (p 3229) | Lichenifications | Folds | Marked pruritus with excoriations In high truing children |
| Pediculosis (p 3185) | Excoriations | Scalp and pubis | Identify nits and lice (crabs) |
| Prurigo mitis (p 3343) | Papules and wheals | Extensor surfaces | Identify offending allergen |
| Purpuras (p 3423) | Ecchymoses telangiectases erythemas wheals or petechiae | Legs and feet | Look for offending allergen often bacterial Get platelet count. Try cevitamic acid |
| Roseola infantum (p 419) | Morbiliform | Generalized | May be mild fever No Koplik spots |
| Scabies (p 3181) | Burrows | Wrists webs of fingers | Identify acarus in burrow |
| Sclerema (p 3157) | Atrophy | Generalized | With rigidity |
| Scurvy (p 3238) | Hemorrhages | Extremities and gums | Vitamin C deficiency Note response to cevitamic acid |
| Subcutaneous fat necrosis (p 3150) | Indurations | Diffusely scattered | Occurs in healthy Spontaneous recovery |
| Syphilis (p 3287) | Macules papules pustules bullae | Palms and soles but may be generalized | Positive serology of mother and/or child |
| Tinea (p 3302) | Scales and crusts | Especially scalp and body | Identify fungus |
| Tuberculosis (p 3258) | Chancre | Penis | After ritual circumcision |
| | Necrotic papules | Extensor surfaces or trunk | Get biopsy |
| | Scrofuloderma | In vicinity of caseous glands | Get biopsy |
| | Lupus vulgaris | Apple jelly nodules on cheeks and nose | Butterfly eruption Get biopsy |
| Urticaria pigmentosa (p 3153) | Macules | Generalized | Wheal on pressure |
| Xeroderma pigmentosum (p 3158) | Macules telangiectases and keratosis | Exposed parts | Pigmentation and photosensitivity |

DIFFERENTIAL DIAGNOSIS OF

Common Dermatoses of Newborn and Infants

| DISTURBANCE | LESION | PREDILECTION | REMARKS |
|---|---|----------------------------|--|
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| Angioma (p 3200) | Strawberry mark, Port wine stain | Nape of neck face and arms | Vascular anomaly |
| Atopic dermatitis (p 3342) | Infantile eczema | Face body and extremities | Seek offending allergen (milk eggs) |
| Auricular fistula (p 3149) | Tract | In front of ear | |
| Contact dermatitis (p 3330) | Moist eczema | Especially diaper area | Distribution of eruption |
| Dermatitis medicamentosa (p 3335) | Maculo-papules erythema | Generalized | History of administration of drug |
| Ectodermal defects (p 3150) | Atrophy | Face hair and nails | Saddle nose |
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| Intertrigo (p 3161) | Acute inflammation | Diaper area | Identify contactual cause |
| Keratosis follicularis (p 3153) | Papules and greasy crusts | Generalized | Vitamin A deficiency |

There is a marked similarity in the appearance of these patients due to the *saddle like depression of the nasal bridge* the *triangularity of the face* as seen in Paget's disease of the bones the thickening and protrusion of the *lips* and the scant growth of very fine silky *hair* on the scalp

No form of therapy is of benefit

EDEMA OF THE NEWBORN (EDEMA NEONATORUM)

Edema of the newborn is a rare condition usually observed in *pre mature marasmic infants*. An edematous swelling is noted in the lower extremities. The swelling then may extend fairly rapidly to involve a large part of the body and is usually accompanied by somnolence and subnormal temperature. The severe cases of this type usually have a fatal outcome due to intercurrent infection. In milder cases the pitting is not so extensive and recovery follows gradual subsidence of the edema.

Treatment consists of maintenance of body temperature and nutrition

EPIDERMOLYSIS BULLOSA

Epidermolysis bullosa is a rare congenital and hereditary disease due to a deficiency or absence of *elastic tissue* in the cutis. In an acquired form in which lesions do not appear until adult life there is said to be no quantitative alteration in the elastic tissue.

Simple and Dystrophic Varieties.—Simple and dystrophic varieties are recognized at birth or soon thereafter. In the *simple type* bullae appear at sites of irritation or injury but there is no involvement of the mucous membranes. The hands and feet are the sites of predilection. The bullae rupture and heal with pigmentation but without scarring or deformity. This form frequently improves or ends in spontaneous recovery at or about puberty and often is associated with latent porphyria and photosensitivity.

The *dystrophic form* is a serious and crippling disease in which there are clear and hemorrhagic bullae over pressure and trauma points. The hands, elbows, knees and feet are sites of predilection but any area may be affected including the buccal mucosa. Severe scarring may result and the nails may be deformed or actually lost. There is interference with locomotion.

Diagnosis.—The disease is readily differentiated from *pemphigus* by the occurrence of bullae only at sites of injury.

Treatment.—There is no satisfactory therapy. The patient must be warned to avoid injury and pressure against the skin. The youthful patient should be guided into an occupation which will shelter him from trauma to upper and lower extremities.

SUBCUTANEOUS FAT NECROSIS OF THE NEWBORN

Subcutaneous fat necrosis of the newborn is a localized disorder which appears in healthy, well-nourished infants from a few days up to three weeks after birth. It differs from *sclerema neonatorum* (p. 3157) in that it is circumscribed, self-limited, occurs in healthy babies and always ends in recovery. The precipitating factor is believed to be *obstetrical trauma*, especially the application of forceps and too enthusiastic resuscitative methods. *Cold* may also play a part in the process.

CUTIS VERTICIS GYRATA

Cutis verticis gyrata is a very rare congenital or acquired disorder which usually appears in adult life. It is seen at times in *acromegaly* but no exact etiologic agent is known.

The occipital region is chiefly affected but the condition may extend over the vertex. It consists of *ridge like linear elevations of the scalp* separated by deep furrows arranged in horizontal or vertical fashion. There is a suggestive resemblance to the convolutions of the brain.

The anomaly is permanent and influenced only by *plastic surgery*.

DERMATOSIS PAPULOSA NIGRA

Dermatosis papulosa nigra is a fairly common *nevus like* (nevroid) condition of *Negroes* affecting females more frequently than males and appearing about the time of puberty. The lesions appear chiefly on the cheeks and eyelids more rarely on the trunk. The eruption consists of multiple

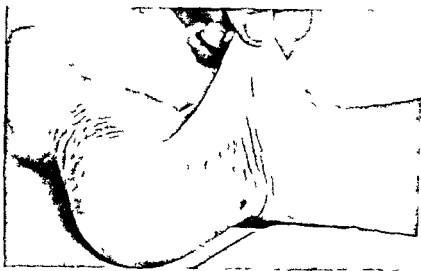


Fig. 888—Cutis hyperelastica

lentil sized rounded soft *papules* which are generally hyperpigmented. They do not ulcerate or degenerate. They resemble pigmented moles, juvenile flat warts and multiple benign cystic epitheliomas.

There is *no satisfactory therapy*. Electrodesiccation leaves depigmented scars which are cosmetically more objectionable than the original lesions.

ECTODERMAL DEFECT

Complete or partial developmental defect of the ectoderm and its appendages may occur as a rare anomaly. The condition affects *males* almost always and is *familial*. It is evidently a developmental anomaly of the germ plasm. There may be absence or maldevelopment of the *teeth*, absence of *body hair*, deformation of the *nails*, few *sebaceous glands* and absence of *sweat glands*.

There is a marked similarity in the appearance of these patients due to the *saddle like depression of the nasal bridge* the *triangularity of the face* as seen in Paget's disease of the bones the thickening and protrusion of the *lips* and the scant growth of very fine silky *hair* on the scalp

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Clinical Manifestations—The condition consists of *areas of induration* in the subcutaneous tissue. These vary from pea to palm size and are *circumscribed well delimited slightly elevated bluish red painless and non tender*. The board like induration, failure to pit on pressure and inability to pinch up the skin are characteristic.

The bluish red color gradually disappears and the areas soften and absorb completely in three to four months generally without any sequelae. The sites of predilection are the *back cheeks arms thighs and buttocks* though other regions (except palms soles and abdomen) may be affected. The general health is unaffected and there are no systemic symptoms.

Histopathology—Pathologically the lesion consists of *necrotic fat cells* and an inflammatory infiltrate of *epithelioid and giant cells*. It has been aptly termed a *lipophagic granuloma* and resembles the picture seen in foreign body granulomas like those produced by injections of camphor in oil, insulin and paraffin (p. 3207).

Course and Treatment—No treatment is required for spontaneous recovery occurs in three to four months.



Fig. 889—Ichthyosis *

ICHTHYOSIS

Several varieties of ichthyosis are recognized by dermatologists. The conditions have in common an alteration in the skin whereby the body covering resembles *fish scales*.

Ichthyosis Simplex—Ichthyosis simplex is a *congenital disease* that is *hereditary and familial* affecting both sexes. It may be present at birth or may not develop for many years.

The condition is easily recognized as the skin is rough and dry and presents a thin brownish fish scale like flaking. There may also be thickening of the skin over the elbows and knees and small keratotic horny plugs (*keratosis pilaris*) on the arms and thighs. These keratotic papules resemble those seen in vitamin A deficiency. The sites of predilection are the extensor aspects of the *arms and legs* but the condition may be more widespread.

Ichthyosis is at its worst in the winter. Excessive use of soap and water aggravates the lesion and induces secondary eczematization.

Ichthyosis Congenita—Ichthyosis congenita (harlequin fetus) is an extremely rare condition in which the infant is usually dead when expelled.

* An Inborn Disease of the Skin

or dies soon after birth. The skin is a hard thickened plate almost shell like. Other gross developmental anomalies are often present.

Congenital ichthyosiform Erythroderma—Congenital ichthyosiform erythroderma is a rare disease in which there first appears a *generalized redness* of the skin. This is followed by *dryness thickening and scaling*. In time the redness may disappear entirely leaving only the ichthyotic condition. The sites of predilection are the flexor surfaces of the extremities and the trunk. In exceptional instances bullae have been observed.

Treatment—Large doses of vitamin A (p 690) are to be administered but little permanent benefit is to be anticipated from any type of therapy.

KERATOSIS FOLLICULARIS (DARIER'S DISEASE)

Darier's disease is a rare cutaneous condition appearing in infancy or childhood and having a definite familial tendency. It occurs in both sexes although somewhat more frequently in males.

Clinical Manifestations—The eruption is usually seen on the scalp, face, neck, the middle of the back, chest, hands and groins although a more widespread distribution may be present. The lesions commence as small skin colored papules chiefly follicular in origin. They enlarge to form red, dish brown lesions covered with a greasy brownish or dirty gray crust. In the folds they become more inflammatory, vegetate and often secrete a thin foul smelling discharge. Vegetation is particularly frequent in the postauricular, axillary and genito-anal regions. The scalp is covered with a profusion of greasy scales and crusts. Papular excrescences have been observed on the hard palate.

Course—The condition may involve only a few small areas or it may spread and become almost generalized. While the disease is not serious in its implications it is extremely unpleasant and even disabling.

Treatment—There has been no satisfactory therapy until recent investigations have shown remarkable improvement but not complete cure with massive doses of vitamin A (p 690). A daily dosage of 200,000 units is given over a considerable period of time. Some benefit probably temporary has been seen after roentgen treatment. Locally preparations of sulfur (p 3128) and salicylic acid (p 3126) as in seborrheic dermatitis are of considerable value.

KERATODERMA PALMARIS ET PLANTARIS

Keratoderma palmaris et plantaris is a rare hereditary condition. The palms and soles become diffusely thickened, dry, hard and yellowish. There is impairment of flexibility. The surface usually is smooth but may be pitted or verrucous. A rarer punctate variety has been observed in which multiple discrete hyperkeratotic patches are present in the palms and soles.

Mal de Meleda—An endemic form of this disease widely present in an isolated part of southern Dalmatia is called Mal de Meleda. It is said to be the result of consanguinity over a long period of years.

Treatment—Treatment is not particularly beneficial but improvement may be obtained by roentgen therapy or the use of strong keratolytics such as 20 to 40 per cent salicylic acid in paste or plaster form (p 3126).

Clinical Manifestations—The condition consists of *areas of induration* in the subcutaneous tissue. These vary from pea to palm size and are circumscribed well delineated slightly elevated bluish red painless and non tender. The board like induration, failure to pit on pressure and inability to pinch up the skin are characteristic.

The bluish red color gradually disappears and the areas soften and absorb completely in three to four months generally without any sequelae. The sites of predilection are the *back, cheeks, arms, thighs* and *buttocks* though other regions (except palms, soles and abdomen) may be affected. The general health is unaffected and there are no systemic symptoms.

Histopathology—Pathologically the lesion consists of *necrotic fat cells* and an inflammatory infiltrate of *epithelioid* and *giant cells*. It has been aptly termed a *lipophagic granuloma* and resembles the picture seen in foreign body granulomas like those produced by injections of camphor in oil, insulin and paraffin (p. 3207).

Course and Treatment—No treatment is required for spontaneous recovery occurs in three to four months.



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Ichthyosis Congenita—*Ichthyosis congenita* (*harlequin fetus*) is an extremely rare condition in which the infant is usually dead when expelled.

| | |
|----------------------------------|---|
| Leprosy | Pigmented anesthetic macules and nodules Get biopsy Stain for M leprae (p 274) |
| Lichen planus | Multiple violaceous papules with central umbilication Associated with intense itching Usually involves flexural surfaces of wrists forearms and legs May be mucosal on tongue buccal mucous membrane and genitals (p 3387) |
| Lupus vulgaris | Apple jelly papules of middle third of face A tubercloid. Get biopsy (p 3265) |
| Melanocarcinoma | Isolated black areas of high grade of malignancy Get biopsy (p 3225) |
| Moles (Nevi) | May be brown, tan, blue or black May be hairy or non hairy If irritated, mole should be excised (p 3204) |
| Mongolian spots | Blue patches over lower back and sacrum Associated with mental deficiency and slit like formations of lids (p 1165) |
| Multiple neurofibromatosis | von Recklinghausen's disease Associated with multiple tumors and pigmented skin areas Cafe-au lait spots |
| Necrobiosis lipoidica diabetorum | Nodular yellow infiltrations on anterior aspects of legs of diabetics May be a hypersensitivity to insulin (p 3240) |
| Pediculosis | May cause brown pigmentation from prolonged scratching Maculae cerulae (blue spots) after prolonged or repeated infestation (p 3182) |
| Pellagra | Vitamin B deficiency With pigmentation of exposed surfaces glossitis and peripheral neuritis Note therapeutic response to vitamin B complex (p 3238) |
| Pemphigus | Fatal dermatosis Characterized by bullae of skin and mucous membranes intense itching and brown pigmentation (p 3405) |
| Perfume dermatitis | Brown pigmentation of face or behind ears due to photosensitivity associated with ingredients of perfume (p 3177) |
| Pinta | A treponematosis with pigmented and depigmented areas of extremities especially palms and soles Note therapeutic response to penicillin (p 353) |
| Pityriasis rosea | Generalized eruption of pink macules so accompanied by intense itching and scaling Benign dermatosis particularly involving trunk and extremities (p 3411) |
| Pityriasis rubra pilaris | Pink papules surrounding hair follicles Note nutmeg feel and black cored papules of dorsum of hand Probably related to deficiency of vitamin A (p 3412) |
| Psoriasis atrophicans vasculare | Brown papules associated with atrophy of the skin and telangiectases (p 3413) |
| Pruritus | Brown pigmentation following all prolonged itching disorders (p 3170) |

CONTINUED

DIFFERENTIAL DIAGNOSIS OF

Localized Pigmented Dermatoses

See also Generalized Pigmentation p 3242

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|---|---|
| <i>Acanthosis nigricans</i> | Pigmentation and papillary excrescences in axillae and other regions Associated with intraabdominal malignancy (p 3355) |
| <i>Angiomas</i> | Strawberry mark Port wine stain Vivid red angiomas (p 3200) |
| <i>Chemical stains</i> | Due to iodine mercurial antiseptics picric acid potassium permanganate coal dust gun powder etc |
| <i>Chloasma</i> | The mask of pregnancy Brown pigmentation of face nipples and areolae (p 2625) |
| <i>Chromophytosis</i> | Pink or dark red patches due to fungous infection Isolate and identify pathogen (p 3300) |
| <i>Cutaneous horn</i> | Yellow to brown or black elevated tumors A precancerosis Biopsy (p 3217) |
| <i>Dermatitis hemostatica</i> | Brown to black pigmentation surrounding ulcerated or healed varicose ulcer of leg (p 3371) |
| <i>Dermatitis herpetiformis</i> | Bilateral and symmetrical groups of vesicles and bullae accompanied by intense itching and burning Usually situated over scapula with residual pigmentation after healing (p 3371) |
| <i>Dermatosis papulosa nigra</i> | Pigmented papules appearing in middle third of face of negro women A congenital disorder (p 3150) |
| <i>Drugs</i> | Brown or black macules with fixed eruptions due to phenolphthalein, barbiturates or antipyrin |
| <i>Ecthyma</i> | Brown pigmentation of body in vagabonds disease Due to combination of pediculosis prolonged scratching and neglected hygiene (p 3252) |
| <i>Erythema ab igne</i> | Brown pigmentation due to prolonged exposure to heat Usually involves shins and arms in workers exposed to heat in industry Often of abdomen in women addicted to hot water bottles and heating pads (p 3169) |
| <i>Epidermolysis bullosa</i> | Congenital disorder in which bullae develop at sites of trauma Healing associated with pigmentation (p 3151) |
| <i>Favus</i> | Fungous disease of scalp with yellow cup-shaped crusts Isolate pathogen (p 3304) |
| <i>Freckles</i> | Tan and brown spots usually on exposed surfaces |
| <i>Friction</i> | Brown pigmentation at sites of constant pressure such as belt region Following prolonged scratching as in pediculosis and lichenifications |
| <i>Idiopathic multiple hemorrhagic sarcomas</i> | Kaposi's disease with multiple purplish nodules on feet and ankles A malignant dermatosis of elderly males Biopsy (p 3226) |

Xanthomas

Disseminated yellow or brown nodules tumors or plaques. Usually associated with profound disturbances of lipid metabolism as in diabetes mellitus and the systemic reticulo endotheloses. Look for glycosuria, hyperglycemia and alterations in cholesterol metabolism (p. 3244).

Xeroderma pigmentosum

Pigmented macules resulting from congenital abnormality producing photo sensitivity of exposed parts. May be associated with telangiectases and keratoses. The latter often become malignant. Get biopsy and protect from actinic and solar rays (p. 3158).

LYMPHEDEMA OF THE LEGS (MILROY'S DISEASE)

Milroy's disease is a rare *hereditary* condition which has a definite *familial* trend and is characterized by the presence of a persistent solid non-inflammatory *edema of the lower extremities*. It usually extends no higher than the knees. The affliction may be noted soon after birth, at puberty or later. There are no evidences of arterial or venous obstruction and there are no collateral signs or symptoms.

There is no fundamental therapy, but *elastic bandages* give symptomatic benefit.

MULTIPLE BENIGN CYSTIC EUTHYROIDISM

See p. 3208

MULTIPLE NEUROFIBROMATOSIS (VON RECKLINGHAUSEN'S DISEASE)

See p. 3206

SCLEREMA OF THE NEWBORN (SCLEREMA NEONATORUM)

Sclerema of the newborn is an extremely serious but fortunately rare abnormality encountered in marasmic infants. The disturbance is characterized by a smooth whitish *shrunken appearance of the skin* which is cold and cadaveric. The process usually *begins in the lower extremities* and rapidly extends to involve the *entire body* with the exception of the palms, soles and scrotum.

The disease pursues a progressively unfavorable course. The body temperature is subnormal. Pulse and respiration become feebler and death invariably follows within a few days.

At autopsy these infants show a hardness of the subcutaneous fat and almost total dehydration. Deposits of fat crystals may be present but necrosis of the adipose tissue is not an accompaniment. The chemical disturbance believed to be responsible for this unfortunate condition is a reduction of the *olein* content of the subcutaneous fat, allowing it to solidify at normal body temperature.

The *treatment* of sclerema neonatorum is usually ineffective. Symptomatic efforts include the maintenance of body temperature and the injection of *dextrose solutions* subcutaneously and intravenously.

See p. 3200

SYRINGOCYSTOMA

| | |
|--------------------------------|---|
| Psooriasis | Chronic and recurrent dermatosis with oyster shell scaling papules. Mostly involving extensor surfaces and flexural areas. Pigmentation usually due to use of chrysarobin as therapeutic application (p. 3414) |
| Radiodermatitis | Brown to black pigmentation following exposure to x rays or radium (p. 3179) |
| Reticulo-endotheliosis | Yellow to brown nodules and plaques associated with profound disturbances of lipid metabolism as in Gaucher's disease and Niemann-Pick syndrome. Note associated hepatosplenomegaly. Identify pathognomonic cells by marrow smear (p. 1137) |
| Riehl's melanosis | Brown to black pigmentations due to exposure to chemicals in industry. Probably a hypersensitivity (p. 3176) |
| Sarcoidosis | Pigmented nodules with telangiectases as manifestation of cutaneous tuberculosis. Associated with parotitis and uveitis. Get biopsy (p. 3271) |
| Sebaceous adenoma | Yellow papules of face. A hereditary disorder associated with tuberous sclerosis causing mental deficiency and other neurologic manifestations (p. 3148) |
| Senile keratosis | Brown scaly areas of exposed parts. A precancerosis. Get biopsy (p. 3216) |
| Seborrheal keratosis | Pigmented scaling warts. A precancerosis. Get biopsy (p. 3217) |
| Solar dermatitis | Deep pigmentation due to prolonged exposure to sunlight (p. 3174) |
| Syringocystoma | Multiple small nodules of anterior trunk. In females (p. 3209) |
| Steatocystoma multiplex | Multiple skin-colored tumors of anterior trunk (p. 3207) |
| Syphilis | Pigmented macules and papules in secondary phase of disease. May be copper-colored. Positive dark field (p. 45). Brown to black pigmented scars after healing. Areas of depigmentation (leukoderma colli). Get serologic tests in addition to local dark field (p. 330) |
| Tinea imbricata | Tropical fungous infection with brownish scaly lesions producing concentric rings. Isolate pathogen (p. 3307) |
| Tinea versicolor | Superficial fungous infection producing multiple pigmented areas. Isolate pathogen (p. 3300) |
| Tattoo marks | Artificial pigmentations produced by dyes such as red sulfide of mercury. Useful in the treatment of localized areas of itching such as pruritus ani (p. 3415) |
| Urticaria pigmentosa | Congenital dermatosis in which pigmented macules wheal when rubbed. Look for possible presence of allergens producing hypersensitivity (p. 3158) |

Xanthomas

Disseminated yellow or brown nodules tumors or plaques. Usually associated with profound disturbances of lipid metabolism as in diabetes mellitus and the systemic reticulo-endothelioses. Look for glycosuria, hyperglycemia and alterations in cholesterol metabolism (p. 3244).

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SYRINGOCYSTOMA

See p. 3209

URTICARIA PIGMENTOSA

Urticaria pigmentosa is an uncommon disease which appears in in fancy childhood or adult life It occurs in both sexes probably with greater frequency in males

Clinical Manifestations—The eruption consists of many brown pigmented *macules* varying in diameter from a small fraction of an inch up to one half inch They are fairly uniform rounded or oval in contour and are in greatest profusion on the *trunk* Lesions may be present on the face extremities palms soles and even in the mouth When a pigmented macule is *rubbed* it becomes elevated and somewhat reddened, resembling a wheal *Itching* may be present but is more frequently absent There may be an associated *dermographism*

Histology and Diagnosis—The characteristic finding in the histologic examination is an unusual abundance of *mast cells* in the corium Differential diagnosis from other pigmentations (p 3154) is made by the history the pathognomonic urtication on friction and the distinctive histopathology

Course and Treatment—The disease reaches its maximum distribution after a period of years and then ceases its activity although the residual pigmentation may be permanent *Treatment* is ineffectual When there is annoying spontaneous urtication with itching investigation may reveal a specific *allergen* removal of which is followed by relief of the symptom

XERODERMA PIGMENTOSUM

Xeroderma pigmentosum is a rare *hereditary* disease often present in more than one member of the family The etiology is unknown but consanguinity is believed to predispose to the condition The condition usually manifests itself in the first few years of life although its appearance may be retarded for many years

Clinical Manifestations—The skin changes are due to *photosensitivity* an abnormal susceptibility to certain wave lengths of the solar spectrum The exposed surfaces are affected notably the *face neck upper chest hands and forearms* The skin becomes freckled the pigmented *macules* being tan to deep brown and of varying size There are also white atrophic spots telangiectases and raised warty keratoses These *keratoses* very frequently degenerate into basal or squamous cell epitheliomas (p 3223) The cornea may be similarly damaged leading to *keratitis* and opacities with impairment of vision

The condition is easily recognized and bears a striking resemblance to the skin changes of chronic *actinic dermatitis* (p 3175) and chronic *radio dermatitis* (p 3179) both of which are also pre-cancerous

Treatment—Treatment consists of *protection of the patient from light* by the wearing of a hat veil and gloves and the use of light protective topical applications (p 3140) Keratoses are removed by *excision* or *electro desiccation* before they become malignant Treatment with roentgen ray or radium may precipitate malignancy The ultimate prognosis is poor but but with careful attention the patient may be kept alive for many years



Fig 890 —Cutis verticis gyrata



Fig 891 —Erysipelas bullosa



Fig 892.—Dyskeratosis follicularis (Darier)



Fig 893.—Urticaria pigmentosa



Fig 894 — Napkin or diaper dermatitis



Fig 895 — Keloid



Fig 896 — Lichen simplex



Fig 897 — Chublain (pernio)



Fig 898 — Radodermatitis of face and of breast

CHAPTER 144

THE TEGUMENTARY SYSTEM TRAUMA PARASITIC INFESTATIONS AND ANIMAL BITES

| | |
|-------------------------------|--|
| Mechanical Injuries | Injuries Due to Heat |
| Chafing (Erythema Intertrigo) | Chronic Dermatitis Due to Heat |
| Friction Burn | Sudamen Miliaria Crystallina |
| Friction Blister | Miliaria (Miliaria Rubra Eczema Heat) |
| Hyperkeratoses | Urticaria ab Igne |
| Scars and Keloids | Injuries Due to Cold |
| Bedsore | Erysipelas Hiemalis (Winter Itch) |
| Lichen Simplex | Chapping |
| Malignancy | Cutis Marmorata (Marbled Skin) |
| Chemical Injuries | Chilblain and Frostbite |
| | Urticaria Hiemalis |
| | Injuries Due to Actinic Rays |
| | Injuries Due to Roentgen Rays and Radium |
| | Parasitic Infestations and Animal Bites |

This chapter on Minor Surgery (p 3909) is concerned in large part with the traumas inflicted upon the vulnerable cutaneous structures. The dermatologist includes in his special field injuries of the skin resulting from mechanical chemical thermal and radiant energies.

MECHANICAL INJURIES

For the most part the mechanical injuries in the province of the dermatologist are frictional in origin. They include chafing (intertrigo) friction burn friction blister hyperkeratoses (corn and callus) scars and keloids decubitus ulcer (bedsore) lichen simplex and malignancy.

CHAFING (ERYTHEMA INTERTRIGO)

Chafing or intertrigo occurs on opposing skin surfaces exposed to continued friction. The eruption results from the maceration of the superficial layers of the epidermis by the admixture of sweat and other secretions. Intertrigo is encountered most frequently on the delicate skin of infants and in obese individuals with bulging and redundant cutaneous structures. Despite its simplicity and seeming unimportance chafing becomes of significance as a frequent precursor and accompaniment of the *dermatomycoses* (p 3293) and the *pyodermas* (p 3248).

The areas of predilection for intertrigo include the spaces between the fingers and toes the groin the crural region the area between the nates and thighs beneath pendulous breasts and under an apron panniculus and in the axillae. In infants it is often seen in the folds of the neck.

Predisposing Factors.—Intertrigo occurs more commonly in summer when skin secretions are increased and in patients who normally perspire freely. Clothing has much to do with intertrigo. The disturbance is produced and aggravated by tight fitting garments the wearing of woollens next to the skin infrequent bathing inadequate drying after the bath.



Fig 894—Napkin or diaper dermatitis



Fig 89 —Keloid

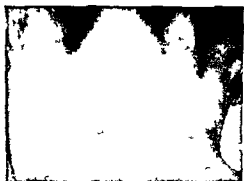


Fig 896—Lichen simplex.



Fig 897—Chilblain (pernio)



Fig 898—Radiodermatitis of face and of breast

| | |
|--|--|
| Lupus erythematosus | Dusky red edematous les on usually involving bridge of nose and cheeks May produce butterfly eruption. Marked photosensitivity Get biopsy (p 3397) |
| Lupus vulgaris | Apple jelly nodules Usually of face A tuberculid Get biopsy (p 3263) |
| Pellagra | Deficiency of nicotinic acid. Productive of edema redness desquamation and hyperpigmentation of exposed surfaces Associated with glossitis Note therapeutic response to vitamin B complex (p 3237) |
| Perfume dermatitis | Erythema and pigmentation of area to which perfume has been applied Often seen at side of neck Due to sensitivity to ultraviolet conditioned by oil of bergamot |
| Radiodermatitis | Following x ray or radium therapy (p 3160) |
| Rosacea like tuberculid of Lewandowsky | Diffuse erythema with brown papules of face in women A tuberculid. Get biopsy (p 3270) |
| Scrofuloderma | Diffuse redness and thickening of the skin Usually overlies a tuberculous lymphadenitis Get biopsy (p 3262) |
| Seborrheal dermatitis | Reddened scalp areas associated with dry or oily scalp (p 3425) |
| Solar dermatitis | Sunburn (p 3174) |
| Streptococcemia | Transitory isolated erythematous eruptions May give blanching reaction with streptococcus antitoxin (p 163) |

particularly in the region between the toes sleeping with excessive bed covers in overheated rooms wearing elastic or rubber accessories such as corsets brassieres suspensories and stockings and the use of coarse rough napkins and diapers

Cutaneous Manifestations—The eruption of intertrigo appears first as a simple redness accompanied by a painful burning sensation of varying degree With further friction and admixture with sweat maceration occurs and the skin becomes more intensely reddened desquamated and edematous Each movement produces considerable discomfort If the condition is neglected secondary fungus or pyogenic infection often complicates the clinical picture

Napkin dermatitis is a special form of intertrigo in infancy It occurs on the skin surfaces exposed to wet diapers The eruption consists of erythematous macules papules vesicles and small ulcerations The condition is due to irritation of the skin by ammoniacal products resulting from the bacterial decomposition of urine in the diapers

Diagnosis—The diagnosis of chafing presents no clinical problem It is necessary however to ascertain whether there are added complicating factors such as *dermatophytosis* (p 3293) or *contact dermatitis* (p 3330) The clinical test furnishes the most satisfactory differentiation Uncomplicated intertrigo responds readily to simple treatment Persistence of the dermatosis or recurrence calls for an investigation for a possible cutaneous irritant (p 3160) or a search for the presence of a superimposed fungus infection (p 3293)

DIFFERENTIAL DIAGNOSIS OF

Local Erythematous Eruptions

See also Generalized Erythematous or Scarletiform Rashes p 180

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|--------------------------|---|
| Acne rosacea | Persistent redness telangiectases and acne form lesions of the face (p 3357) |
| Actinic dermatitis | From exposure to ultraviolet radiation |
| Acrodynia | Pink disease of newborn Redness swelling and pain of hands and feet Probably a vitamin B deficiency Note therapeutic response to vitamin B complex (p 3145) |
| Chilblain and frostbite | Usually of fingers toes nose or earlobes Pain and redness may be followed by vesiculation and gangrene Try anticoagulants (p 3173) |
| Dermatitis hemostatica | Of legs with varicose vein complex. Usually followed by ulceration (p 3371) |
| Dermatitis medicamentosa | Erythematous eruptions following local or systemic administration of therapeutic substances to those with hypersensitivity (p 3335) |
| Erythema multiforme | Toxic dermatosis with variegated lesions Seek offending allergen (p 3375) |
| Erythromelalgia | Vasomotor neurosis Episodes of pain, redness and swelling of extremities (p 1002) |
| Exfoliative dermatitis | A toxic dermatosis Often due to therapeutic substances such as arsenic sulfonamides and gold Followed by scaling of the skin Often fatal (p 3385) |
| Erysipelas | St Anthony's Fire Local streptococcal lesion with fiery red advancing border Often associated with constitutional symptoms (p 167) |
| Erysipeloid | Reddened inflammatory lesions of the hands in fish handlers (p 328) |
| Erythema ab igne | Redness due to local application of heat (p 3169) |
| Gout | Painful red swelling Usually of great toe with distention of superficial veins Check blood uric acid Note therapeutic response to colchicine (p 2867) |
| Granuloma fungoides | Fatal dermatosis with itching and redness of the skin, followed by the formation of nodules plaques and fungating tumors Get biopsy (p 3385) |
| Friction burn | At site of local pressure |
| Granulosis rubra nasi | Redness of the nose Usually seen in children Associated with marked sweating (p 3464) |
| Contact dermatitis | From local exposure to irritants in the hypersensitive (p 3330) |
| Intertrigo | Chafing of opposing skin surfaces Usually seen under the breasts between the legs and in the axilla (p 3161) |
| Keloid | Fibrous tissue overgrowths producing hypertrophic scar (p 3160) |

this way. It is best to express the contents of the vesicle by pricking the edge with a sterile needle draining the contents and applying a protective dressing using the roof of the blister to cover the underlying and denuded epithelium. If there is the slightest suspicion or danger of infection the roof of the blister is removed and the denuded surface is treated with *penicillin ointment* (p 3124).

HYPERKERATOSES

Prolonged friction produces hyperkeratosis resulting in the appearance of a *corn* (*clavus*) or a *callus* (*callositas*).

The Corn (Clavus).—The corn is a small horny growth commonly observed on the toes. It is situated in the epidermis but presses down upon the corium. It consists of a central conical core whose widest part is external and whose narrow apical portion is deeply situated. The apex makes pressure upon nerves causing exquisite pain. Corns result from intermittent pressure over bony prominences. The most frequent cause is a tight or poorly fitted shoe. The growth is apparently an attempt on the part of the skin to protect itself from injury.

Two varieties of corn are encountered: the *hard corn* which appears on the sole or the outer surfaces of the toes and the *soft corn* which is usually present between the toes. The hard corn is smooth, shiny and slightly elevated. The soft corn is soggy and grayish white due to the heat and moisture of the interdigital space.

Treatment requires the wearing of properly fitted shoes. In some instances this measure alone is followed by disappearance of the corn. After prolonged soaking of the feet in hot water the corn may be removed by paring and coring with a sharp knife or by the application of strong *keratolytics* (p 3136). Favored preparations are 10 to 20 per cent *salicylic acid* in collodion or 20 to 40 per cent *salicylic acid* plaster. These are re-applied for several nights after which the corn can often be picked out en masse. With recurrence despite proper therapy an orthopedic surgeon should be consulted about possible underlying bony pathology such as an exostosis or a hammer toe which may require operative treatment.

Obstinate lesions may respond to *roentgen* therapy.

The Callus (Callositas).—A callus is a hard thickened area of skin a consequence of hyperplasia of the horny layer encountered most often upon the palms and soles. It occurs as a firm skin colored yellowish or grayish patch and is more elevated centrally than at the margin. It tends to blend without distinct border into the normal skin.

The callosity results from continued pressure and like the corn represents a protective effort of the skin. Calluses on the ball of the foot are caused by badly fitted shoes and/or prolonged standing. They may also appear as occupational traumas. On the palms they are seen in laborers, oarsmen, blacksmiths and carpenters. In harpists and violinists similar changes, not so severe, are seen on the terminal phalanges. In trombonists they appear on the lips.

Treatment may be unsuccessful if the occupation is continued. However, considerable relief is obtained by prolonged soaking in hot water followed by paring with a sharp knife. *Keratolytics* (p 3136) are also useful as adjuvants provided that the irritation does not interfere with function.

The lesion of napkin dermatitis may resemble the moist papular lesions of *secondary syphilis*. In the latter condition other lesions of active pre-natal syphilis are usually present. Darkfield examination (p 45) shows the presence of the spirochete and the Wassermann test of the blood is positive. *Seborrheic dermatitis* affects the folds more sharply and is present in other areas. *Contact dermatitis* may be differentiated by failure to respond to simple therapy.

Treatment—The treatment of intertrigo requires first that the irritant be removed. Clothing, corsets, brassieres, shoes, socks, trusses, suspensories, elastic stockings, girdles and night clothes should be examined as possible etiologic agents. Suggestions should be made as to changes that will prevent further trauma. It may be necessary to get along for a few days without an unessential accessory such as a truss or a corset. In infants with napkin dermatitis the mother is instructed not to permit the child to be in prolonged contact with wet diapers. The use of rubber panties is particularly harmful. Nylon substitutes may produce a contact dermatitis of chemical origin.

When wide areas of skin surface are denuded and there is considerable exudation and edema, complete *bed rest* is desirable until the lesion dries and epithelization occurs. Opposing skin surfaces are separated by carefully interposing widths of nonabsorbent cotton or lamb's wool. *Albolene*, olive oil or cotton seed oil are freely applied to lubricate the surfaces. Sprinkling the area with *talcum powder* protects denuded surfaces provided that the excess is wiped off and not permitted to cake in the folds and creases. Moist lesions are best treated with cooling *wet dressings* (p 3134) or with the simple oils, since ointments and talcum tend to add to the discomfort. The use of *soap* and *water* may also prove irritant. It is more comforting to paint on a simple lotion of *calamine* (p 3115) several times daily.

After the relief of the dermatosis the practitioner guards against *recurrence* by eliminating or correcting the hygienic error. *Obese patients* are encouraged to go on a reduction diet (p 697). Proper clothing and bathing habits and well fitting accessories are recommended. The parts are to be kept dry and eased from friction by the liberal use of a simple dusting powder.

FRICION BURN

The friction burn consists of a simple *erythema*. Unless the superficial layers of the skin are denuded or destroyed the cutaneous disturbance disappears shortly after the discontinuance of the trauma.

The *treatment* of the friction burn requires no unusual care other than a prevention of repetition of the difficulty. The use of a *protective powder* (3137) or an *emollient* is often of assistance.

FRICION BLISTER

The friction blister results when there is an effusion of *serum* between the layers of the skin.

The *treatment* of the blister primarily requires *protection* against the introduction of infection by using a protective ointment and dressing. On a vulnerable surface such as the palm the blister cannot be handled in

The occurrence of *malignant degeneration* in scars is not infrequent. It is most commonly observed in burn scars many years after the lesion was incurred. The *carcinoma* is of the squamous cell variety and while not highly malignant may cause a fatal outcome if not diagnosed early and treated properly.

Scars cannot be removed but the *plastic surgeon* (p 3993) may *revise the lesion* and *correct disfigurement* and *disability*.

Keloids—A keloid is an overgrowth of fibrous tissue having the nature of a hypertrophic scar. Keloids develop after injury to certain skins and also in the healing of some morbid processes. They occur constantly in *dermatitis papillaris capilliti* (p 3255). Keloids may follow the healing of burns, zoster, acne vulgaris and syphilitic lesions. They may result from very slight traumas as squeezing a pimple, an insect bite, piercing the ear lobes and even scratching. The reaction is most frequently observed in the Negro race.

Keloids may be single or multiple, small or enormous. They develop slowly and at first are *reddish*, *itching* and *tender elevations*. In time they become *white* and *insensitive*. They are firm, flattened or rounded and of rubbery consistency. While lesions may occur in any portion of the skin surface, they are most frequent on the *anterior chest wall* and the *face, neck, ears* and *extremities*.

The most successful therapeutic measure is the application of *roentgen rays* or *radium* (p 3796). Radiation is most effective in the early stage of development and often yields an excellent cosmetic result. In old keloids, radiation therapy may be completely without benefit and the best procedure is *excision* of the lesion and the use of *radiation therapy* in the immediate postoperative period to prevent recurrence.

BEDSORES (DECUBITUS ULCERS)

Bedsore or decubitus ulcer occurs over prominent bony points subjected to prolonged pressure. They are particularly observed in *chronic illness* when the patient has been confined to bed for a long period. Malnutrition, impaired circulation of the tissues, disease of the spinal cord and prolonged application of tight or ill fitting plaster casts act as predisposing factors.

While the lesions are most common over the sacrum, they also occur on the heel and elbow. The first change is a permanent dull red discoloration after which the tissues break down and form indolent unhealthy ulcers.

Treatment—Of the greatest importance is the *prevention* of bed sore formation in bedridden patients. *Air cushions* and *cotton rings* are used over the pressure points. The skin is hardened by *alcohol rubs* and the application of *Compound Tincture of Benzoin* (p 3114). Friction is diminished by oiling and dusting with *talc*. Change of position is mandatory at frequent intervals. Soiling with urine, feces or secretions must be prevented.

When ulceration is already present, the lesion is treated with a mixture of equal parts of *Castor Oil* and *Balsam of Peru*. *Lassar's Paste* or *Ointment of Zinc Oxide*. *Silver Nitrate* application is used to stimulate

SCARS (CICATRICES) AND KELOIDS

Scars and keloids represent a reaction at the site of an injury an incision or a disease process

 DIFFERENTIAL DIAGNOSIS OF

Keratoses

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|---------------------------|---|
| Actinic dermatitis | Following prolonged exposure to solar or ultraviolet rays |
| Arsenical keratosis | On palms and soles As the result of administration of inorganic arsenic A precancerosis (p 3216) |
| Callositas | Calluses from prolonged pressure Usually on palms and soles (p 3168) |
| Clavus | Corns Usually over bony prominences or on soles as the result of ill fitting shoes |
| Chemical injuries | Following the use of irritants in occupations Particularly associated with exposure to tar |
| Keratoderma climactericum | Fissures and keratosis of palms and soles at menopause (p 3240) |
| Occupational keratosis | Precancerosis in workers exposed to tar tar distillates pitch aniline dyes soot, petroleum and machine oil (p 3169) |
| Radiodermatitis | Atrophic and hyperkeratotic changes with precancerous tendency History of treatment with x ray or radium (p 3179) |
| Onychomycosis | Ringworm of nails with hyperkeratosis in the subungual regions (p 3455) |
| Pinta | Treponematoses with keratosis of palms and pigmentary disturbances Note therapeutic response to penicillin (p 353) |
| Radiodermatitis | With areas of atrophy and telangiectases History of treatment with radium or x rays (p 3179) |
| Seborrheal keratosis | Multiple pigmented scaly warts A precancerosis (p 3217) |
| Senile keratosis | Rough scaly lesions of exposed parts A precancerosis Get biopsy (p 3216) |
| Vitamin A deficiency | Producing keratosis pilaris with papules surrounding mouths of hair follicles Note therapeutic response to vitamin A |
| Xeroderma pigmentosum | Congenital disorder with pigmented macules atrophy telangiectases keratosis and abnormal photosensitivity A precancerosis Get biopsy (p 3158) |

Scars —Scars may be of many shapes and sizes The fresh scar is red dish and tender later it becomes white and insensitive Scars which result from extensive burns are often contracted and may produce marked deformity about the face neck and joints

Weak acids and alkalis often cause a dermatitis characterized by erythema vesiculation and scaling. Housewives dishwashers and laundry workers often have chronic dermatitis from the alkali in soap and soap powders. The *stronger acids and alkalis* such as sulfuric acid and lye are more likely to produce burns which if severe become ulcerated. The metals and metallic salts more often induce dermatitis with indolent ulceration. Tar pitch and petroleum distillates in addition to dermatitis produce photosensitization with *secondary skin changes folliculitis pyoderma* and acne-like eruptions of the face and extremities. In long standing examples keratoses may occur and these occasionally undergo malignant degeneration.

Treatment—The management of occupational dermatoses is begun by the recognition of the *etiologic agent* and attempts to *eliminate exposure* to it. Patients who must continue at an occupation should be instructed as to methods of protecting the hands with gloves or rapidly removing the irritant by frequent washing. The presence of *keratoses* and suggestive *malignant degeneration* is sufficient reason for barring the sufferer from the industry.

The lesions themselves are treated according to local indications. An acute dermatitis is soothed by protective *wet dressings* (p 3134) *lotions* (p 3137) and *liniments* (p 3135). Chronic dermatoses often respond to stimulation with *tar ointments and pastes* (p 3131). *Roentgen therapy* (p 3796) is invaluable in the more resistant forms.

INJURIES DUE TO HEAT

Cutaneous injuries due to heat are caused in a number of different ways. *Acute burns and scalds* are more properly discussed in the chapter on Minor Surgery (p 3909). The interest of the dermatologist is more often directed to the less dramatic caloric manifestations such as *chronic dermatitis from heat* (erythema ab igne) *sudamen miliaria rubra* (prickly heat) and *urticaria ab igne*.

CHRONIC DERMATITIS DUE TO HEAT (ERYTHEMA AB IGNE)

Prolonged intermittent exposure to dry heat occurs among persons who like to toast their shins in front of an open fire or a radiator and those whose work exposes them to great heat (coke stokers cooks engineers). It may also result from the protracted use of a hot water bottle over any part of the body especially the abdomen. The dermatitis consists of a reticulated mesh like area of erythema which subsequently assumes a brownish tint. It may persist for many years after its inception.

Treatment of course consists in avoidance of exposure.

SUDAMEN (MILIARIA CRYSTALLINA)

The sudaminal lesion is caused by a blockage of the outlet of the *sweat duct*. As a result there are formed multiple small clear noninflammatory crystalline *vesicles* whose contents are pure *sweat*. The eruption appears suddenly and is usually confined to the *trunk* where it is invariably multiple. Sudamen is generally transitory and is seen in febrile and debilitated patients.

Treatment consists of *alcohol sponges* and the liberal use of *talcum powder*.

epithelial growth Gangrenous sloughs are removed with scissors and forceps Applications of gauze soaked with 2 to 10 per cent pectin powder (p 3123) mucilage of tragacanth or tragacanth paste (p 3131) are highly successful These are applied to the ulcer covered with oiled silk or cellophane and held in place with scotch tape When the ulcer is infected sulfathiazole or penicillin ointments (p 3124) may be used

LICHEN SIMPLEX

Lichenification is not a disease but rather an alteration of the skin induced by prolonged moderate trauma The most important causative agency is *scratching* Many cutaneous conditions associated with *pruritus* develop secondary lichenification and this alteration is a constant concomitant of *lichen simplex chronicus circumscriptus* (p 3229) Lichen simplex seldom occurs in stable individuals It is more likely to affect *psycho neurotic* and *neurotic patients* who are nervous irritable and lacking in emotional control

The *lesions* of lichen simplex are characterized by a thickened *leathery appearance* of the skin The surface is composed of flat shiny elevated planes with intervening exaggerated furrows As the result of the itching which accompanies the disease there are usually *secondary excoriations* from the scratching

Treatment consists in the attempt to stabilize the nervous system of the individual patient and to allay itching by means of *antipruritic remedies* (p 3136) particularly lotions and medicated slaves If the itching and eruption persist the patient should be referred to the specialist for *roentgen therapy*

MALIGNANCY

Prolonged friction carried out over periods of years has been known to give rise to malignant change This has been observed most often in primitive countries where man is used as a beast of burden and is virtually unknown in relatively civilized communities where labor and laborers are no longer exploited to this extreme degree

CHEMICAL INJURIES

Direct contact with irritant chemicals produces pathologic changes in the normal skin These lesions are not to be confused with *contact dermatitis* (p 3330) in which the dermatosis is evoked in a relatively small proportion of patients suffering from a state of hypersensitivity

Chemical injuries to the skin most often arise in industry The Office of Dermatoses Investigation of the United States Public Health Service has made an intensive study aimed at the recognition prevention and treatment of the various skin afflictions due to the use of chemicals by workers in industry These are detailed in the chapter on *Toxicological Substances* (p 4000) which also includes a consideration of the effects of *War Gases* (p 745)

Types of Reaction—In general chemical injuries to the skin give rise to a wide variety of pathologic manifestations There may be acute or chronic *dermatitis folliculitis pyoderma ulcers keratoses* and *malignant degeneration*

| | |
|------------------|---|
| Pruritus senilis | Probably an endocrinopathy Note effects of administration of androgen or oestrogen (p 2515) |
| Psychogenic | Most frequently observed form May be particularly localized to erotic areas such as vulva inguinal regions or anus Refer to psychiatrist |
| Serum sickness | Usually associated with intense urticaria Get history of previous injection of biological |
| Uncleanliness | A frequent complaint in the great unwashed Often associated with pediculosis or scabies Look for evidences of infestation on body in wearing apparel and in bedding |

MILIARIA (MILIARIA RUBRA PRICKLY HEAT)

Prickly heat is caused by high temperatures of febrile or atmospheric origin or induced by working in circumstances that expose one to great heat Profuse perspiration obesity and the wearing of heavy nonporous clothing predispose to the eruption which appears rapidly and is most profuse upon the trunk

The *rash* consists of tiny inflammatory papules and vesicles Occasionally there is secondary infection and the formation of tiny pustules The papulovesicular lesion is distinctly red and inflammatory inclined to be widespread and accompanied by itching and burning sensations

Treatment demands the avoidance of excessive heat Appropriate attire should be worn if exposure is inevitable Alcohol and hot drinks are eliminated from the diet The use of soap may irritate the skin Local applications of dusting powder (p 3137) are adequate and 0.25 to 0.5 per cent menthol may be added to relieve pruritus (p 3136) Soothing lotions (p 3137) such as calamine are used when there is moisture or vesiculation

URTICARIA AB IGNE

Urticaria ab igne is a manifestation of allergy (p 3329) arising from a physical source

INJURIES DUE TO COLD

Dermatoses and other afflictions of the skin result from cold following the rigors of winter exposure in industries that deal with refrigeration and from the therapeutic application of dry ice (carbon dioxide snow) Refrigeration therapy (cryotherapy) represents an attempt to reduce the metabolic activities of tissue in the management of amputations and inoperable malignancy (p 3786)

The skin conditions resulting from abnormal cold include pruritus hiemalis (winter itch) chapping cutis marmorata (marbled skin) chilblains frostbite and urticaria hiemalis The effects of cold are also manifest on the deeper tissues in Raynaud's disease (p 1000) and paroxysmal hemoglobinuria (p 1075)

PRURITUS HIEMALIS (WINTER ITCH)

Adults especially elderly persons with dry skins occasionally develop pruritus in cold weather The itching occurs mostly on the legs and

DIFFERENTIAL DIAGNOSIS OF

Generalized Pruritus (Itching)

Itching is the commonest most characteristic and most annoying of the subjective symptoms that originate in the skin. The sensation of itching is perceived and conveyed by the receptors and pathways for pain. The complaint may be so intractable and unbearable that the suffering engendered exceeds sensations of pain. Eventually the dermatosis is complicated by excoriations and crusting.

See also Localized Pruritus (p 3178)
 Pruritus ani (p 3415)
 Pruritus Vulvae (p 2594)

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|-------------------------|---|
| Allergy | Hyper sensitivity to digestants and drugs. May cause pruritus without other cutaneous manifestations. Obtain diary history (p 3474) |
| Bath pruritus | Hypersensitivity to water. Often associated with pruritus senilis. Use colloidal baths (p 3133) |
| Chemical | Hypersensitivity to soap flannels and woollens |
| Drug pruritus | Particularly manifested by the opiates |
| Endocrinopathies | Hyperthyroidism with elevation of BMR and therapeutic response to iodide and Deracil (p 1210). Gonadal disturbances particularly in pregnancy during menstruation at menopause and in the senile (p 3240) |
| Exanthems | In the phases of desquamation particularly in measles and scarlet fever (p 172) |
| Exfoliative dermatitis | With generalized scaling. Often the result of hypersensitivity to therapeutic agents such as arsenic sulfonamides and gold (p 3383) |
| Granuloma fungoides | Mycosis fungoides. A fatal dermatosis with intense itching in pre-eruptive phase. Later note scaling nodules and tumors with ulceration. Get biopsy (p 3386) |
| Helminthiasis | Probably a hypersensitivity to protein of invader. Note eosinophilia. Examine stools for ova and parasites (p 1893) |
| Hematopoietic disorders | Particularly in leukemia and Hodgkin's disease. May also be observed with the anemias and polycythemia. Check hemogram marrow smears and lymph node biopsy if necessary (p 1046) |
| Jaundice | Particularly of obstructive varieties (p 1951). Examine urine stool and serum for bile pigment disturbances (p 1965) |
| Metabolic disorders | Diabetes mellitus gout uremia and cholemia unassociated with jaundice. Note increase in blood sugar uric acid urea and nonprotein nitrogen (p 3713) |
| Pemphigus | Fatal dermatosis with large bullae of skin and mucous membranes. Get biopsy (p 3411) |
| Pruritus hiemalis | Winter itch. Often due to hypersensitivity to clothing bath water or soap (p 3171) |

CHILBLAIN (ERYTHEMA PERNIO) AND FROST BITE
(DERMATITIS CONGELATIONIS)

Chilblain occurs after exposure to severe cold. It is most common in children and aged persons and in those whose tissues are devitalized by *chronic illness or impaired circulation*.

The lesions appear as areas of tensely edematous and elevated *erythema*. They affect primarily terminal portions such as *fingers toes nose and ears* but may also appear on the legs. The lesions feel cold and are sensitive to touch. The patient complains of severe *burning and itching*. This is the simplest form of the condition and prompt treatment practically always causes a return to normal.

More severe changes ensue in *frost bite* because of *circulatory obstruction*. *Vesicles and bullae* appear, areas of *erosion* are noted and *ulceration and gangrene* may result. These severer symptoms arise when the exposure to cold continues beyond the early stages so that occlusive changes occur in the blood vessels. With the threat of gangrene, deposits of heparin (p 1050) are suggested for anticoagulant effects. See Fig 897 p 3160.

Treatment—Treatment requires the *gradual application of warmth* to the affected area, for excessive heat may damage the tissues. In mild cases, gentle *rubbing* may be adequate. *Heavy massage or pressure is to be avoided* as it may destroy tissue. After the circulation has been improved, the skin is anointed with oil and covered with absorbent cotton.

In the more severe cases, warmth should be applied gradually and gently in an effort to restore the circulation. The initial temperature of the water should not exceed 50 to 60° F. The temperature is gradually raised, provided the patient does not complain of pain. If the area is blistered, the bullae are evacuated and a *protective ointment* is applied. In the presence of ulcerations, 2 to 10 per cent *pectin powder* (p 3123) in sterile water may be applied and covered with oiled silk. *Cod liver oil ointment* (p 3117) is also recommended.

To *prevent recurrence* of the disturbance, the patient is warned to avoid excessive cold. If this is impossible, heavy woolen socks and gloves are worn. Silk socks or stockings are used next to the skin and garters, tight gloves and snug fitting shoes are avoided. The skin is inured to cold by *daily immersion in cold water* and *contrast baths* in hot and cold water. If unusual cold is anticipated, a layer of oil serves as an insulator.

URTICARIA HIEMALIS

An urticarial eruption due to exposure to cold is a cutaneous manifestation of *physical allergy* and is elsewhere described (p 3351).

INJURIES DUE TO ACTINIC RAYS

The skin may be damaged by actinic rays derived from *sunlight* or a *therapeutic lamp*. The effect produced depends upon the constitution of the individual skin, the existence of a state of hypersensitivity to light (*photosensitivity*), the *intensity* of the waves and the *duration* of the exposure. The effects of all light waves depend not on their heating qualities but upon their absorption by the cellular cytoplasm which they injure.

thighs but may affect the trunk and upper extremities. As a rule the discomfort is inconstant and most marked in the evening when the patient disrobes. Scratching may lead to cutaneous alterations producing a traumatic dermatitis and in some instances *lichenification* (p 3167). The condition tends spontaneously to abate and disappear with the advent of warm weather.

Treatment consists in avoiding soaps and woolen garments against the skin and in less frequent bathing the use of mineral oil in the bath and the oral administration of *vitamin A* (p 617). Locally, soothing *antipruritic lotions* (p 3138) and *pastes* or *talcum* are applied. Highly recommended is a combination of equal parts of lanolin petrolatum and benzoated lard containing 2 per cent of salicylic acid and $\frac{1}{3}$ to 1 per cent of menthol.

| | |
|-------------------|------------|
| ℞ (Ormsby) | |
| Salicylic Acid | 24 |
| Menthol | 0.4-1.2 |
| Lanolin | |
| Petrolatum | |
| Benzoated Lard | as ad 1000 |
| Ft. Ung. | |
| Sig. External Use | |

CHAPPING

Superficial fissuring of the skin and lips is seen in cold weather the *hands* and *face* most frequently being affected. Dryness of the skin and the frequent use of *harsh soaps* predispose to the condition. On the vermilion surface of the lips the condition is aggravated by exposure to wind and moisture.

To relieve the chapping the skin should be thoroughly *dried* after washing especially before going out into the cold. *Gloves* should be worn when the patient leaves the house. *Mild soaps* preferably of the *sulfonated oil type* (p 3133) are preferred. The skin is protected against the cold and its condition is improved by the use of *cold cream* mixtures of *glycerin* and *rose water* or a *dusting powder*. On the lips *camphor preparations* and a colorless lip pomade in the form of a lipstick are to be used. The patient should be warned against moistening the lips with the tongue.

CUTIS MARMORATA (MARBLE SKIN)

Cutis marmorata is a transitory noninflammatory disorder due to *spasm* of the small cutaneous vessels. It is brought on by exposure to cold weather. There is no discoverable cause and it occurs in persons who are otherwise completely normal. It is more frequently observed in children than adults.

The condition is most often seen on the *legs* which display a reticulated mesh like arrangement of bluish red lines enclosing areas of completely normal skin. The lines are caused by dilated larger blood vessels.

Treatment consists of wearing clothing more protective against the cold.

CHILBLAIN (ERYTHEMA PERNIO) AND FROST BITE
(DERMATITIS CONGELATIONIS)

Chilblain occurs after exposure to severe cold. It is most common in children and aged persons and in those whose tissues are devitalized by *chronic illness or impaired circulation*.

The lesions appear as areas of tensely edematous and elevated *erythema*. They affect primarily terminal portions such as *fingers toes nose and ears* but may also appear on the legs. The lesions feel cold and are sensitive to touch. The patient complains of severe *burning and itching*. This is the simplest form of the condition and prompt treatment practically always causes a return to normal.

More severe changes ensue in *frost bite* because of *circulatory obstruction*. *Vesicles and bullae* appear, areas of *erosion* are noted and *ulceration and gangrene* may result. These severer symptoms arise when the exposure to cold continues beyond the early stages so that occlusive changes occur in the blood vessels. With the threat of gangrene, deposits of heparin (p 1050) are suggested for anticoagulant effects. See Fig 897 p 3160.

Treatment—Treatment requires the *gradual application of warmth* to the affected area. For excessive heat may damage the tissues. In mild cases, *gentle rubbing* may be adequate. *Heavy massage or pressure is to be avoided* as it may destroy tissue. After the circulation has been improved, the skin is anointed with oil and covered with absorbent cotton.

In the more severe cases, warmth should be applied gradually and gently in an effort to restore the circulation. The initial temperature of the water should not exceed 50 to 60° F. The temperature is gradually raised provided the patient does not complain of pain. If the area is blistered, the *bullae* are evacuated and a *protective ointment* is applied. In the presence of ulcerations, 2 to 10 per cent *pectin powder* (p 3123) in sterile water may be applied and covered with oiled silk. *Cod liver oil ointment* (p 3117) is also recommended.

To *prevent recurrence* of the disturbance, the patient is warned to avoid excessive cold. If this is impossible, heavy woolen socks and gloves are worn. Silk socks or stockings are used next to the skin and garters, tight gloves and snug fitting shoes are avoided. The skin is inured to cold by *daily immersion in cold water* and *contrast baths* in hot and cold water. If unusual cold is anticipated, a layer of oil serves as an insulator.

URTICARIA HIEMALIS

An urticarial eruption due to exposure to cold is a cutaneous manifestation of *physical allergy* and is elsewhere described (p 3351).

INJURIES DUE TO ACTINIC RAYS

The skin may be damaged by actinic rays derived from *sunlight* or a *therapeutic lamp*. The effect produced depends upon the constitution of the individual skin, the existence of a state of hypersensitivity to light (*photosensitivity*), the *intensity* of the waves and the *duration* of the exposure. The effects of all light waves depend not on their heating qualities, but upon their absorption by the cellular cytoplasm which they

The cutaneous effects of ultraviolet rays are the same whether the source is ordinary sunlight or a therapeutic lamp. The active portion of the solar spectrum for the skin lies between 2900 and 3130 angstrom units. The most potent rays are those in the neighborhood of 2967 angstrom units. Waves of this length are present with the greatest intensity in therapeutic ultraviolet radiation and less so in natural sunlight.

Variations in Reaction—While ultraviolet rays from an artificial source and sunlight are of benefit in certain cutaneous diseases (p 3794) they may cause *aggravation* of some eruptions and actually *initiate* others (p 3176). The individual variation in the reaction to ultraviolet rays is considerable. The Negro's skin is most resistant while the blond's is least so. Many patients exhibit a state of *photosensitivity* (p 3176) in which there is a susceptibility or hypersensitivity to light so that violent reactions follow slight exposure. Ordinarily the *degree of reaction* varies inversely with the amount of pigment present in the deeper layers of the skin. The thickness of the horny layer also influences the response which is always greatest in those areas where the skin is thinnest.

Actinic Dermatoses—The cutaneous lesions affected or induced by ultraviolet light include acute solar or actinic dermatitis (*sunburn*) chronic actinic dermatitis and ephelides (*freckles*). As manifestations of physical allergy *solar urticaria* and *herpes simplex* occur in the sensitive individual.

In addition to the actual dermatoses that result from exposure to actinic rays certain cutaneous lesions exhibit photosensitivity. These include *hydra aestivale*, *lupus erythematosus* and *xeroderma pigmentosum* (p 3176). Additionally the sun's rays are activating agencies in certain types of *melanosis* and in the pigmentary dermatitis from perfume (*Berlocke*). The latter are described in subsequent paragraphs.

SUNBURN (ACUTE SOLAR DERMATITIS ACUTE ACTINIC DERMATITIS)

Solar dermatitis usually appears after a latent period of two to four or more hours. The initial eruption consists of an *erythema* which if intense progresses to the formation of *vesicles* and *bullae*. There is an associated sensation of warmth, burning and some itching. If only small areas of skin are affected there may be no systemic symptoms but when large portions of the skin surface are involved the patient may have *headache*, *nausea*, *fever*, *restlessness*, *albuminuria* and other signs of toxemia.

Under ordinary circumstances, the condition subsides within a few days and is followed by *desquamation* and *pigmentation* (tanning). The pigmentation is greatest in the brunette and is slight or absent in blonds and red haired persons.

Treatment—*Prevention* of sunburn is accomplished by the avoidance of direct or prolonged exposure to sunlight and the use of preparations which filter out the ultraviolet rays (p 3140). Persons who show a high degree of susceptibility so called *photophobes* had better avoid exposure since serious consequences may result. In most people gradually increasing exposures rather than sudden prolonged ones result in habituation of the skin and increased tolerance of at least temporary duration. Most *oils* and *creams* have some ability to filter out ultraviolet rays but the addition of *quinine*, *salol* or *menthyl salicylate* (p 3140) enhances this effect.

The *treatment* of acute sunburn depends upon the extent

When there are toxic symptoms rest in bed forced fluids and sedation are indicated. The ordinary sunburn requires only local treatment. *Cold compresses* are very grateful and in addition *mentholated talcum powder* and *calamine liniment or lotion* afford considerable relief.

The use of *picric acid* or the local anesthetic ointments is inadvisable because of their tendency occasionally to evoke a severe contact dermatitis.

CHRONIC ACTINIC DERMATITIS (CHRONIC SOLAR DERMATITIS)

Prolonged intermittent exposure to strong sunlight produces chronic changes in the skin. These occur most commonly in persons whose occupation necessitates daily exposure (*farmers sailors*). It is more frequent in blonds and red haired persons. The exposed surfaces are naturally those which reveal the alterations. The dorsum of the hands the face and the vermilion surface of the lips are most affected. The change in the lower lip may be marked (*chronic actinic cheilitis*) leading to scaly atrophic areas keratoses and squamous cell carcinoma.

The eruption is composed of pink or brown *macules* in a skin which is thickened and discolored. Later the skin becomes thin and wrinkled and resembles somewhat the skin of chronic radiodermatitis. In time there develop *scaliness* areas of *atrophy* and circumscribed elevated grayish *keratotic plaques* important because of their tendency to degenerate into *squamous cell carcinoma* (p 3220).

Treatment—As in acute sunburn *preventive measures* by protective clothing and medicaments are recommended. An educational program directed at those whose occupation and geographic location make them candidates for this condition would be of great value.

FRECKLES (PHELYDES)

Freckles are yellow or brownish *macules* which appear most commonly on the face but also upon the covered portions of the body. They may be few or so numerous as to almost cover the face. Freckles are most common in children young adults blonds and red haired individuals. They tend to become darker and more numerous in summer and to fade in winter.

Freckles have been variously regarded as abnormal collections of basal cells containing large amounts of pigment or congenital birth marks or nevi. They are unimportant except as cosmetic blemishes and are not capable of malignant degeneration.

Treatment—In predisposed individuals prevention should be practiced by avoiding strong sunlight or ultraviolet light and by the local use of *light filtering medicaments* (p 3140). The freckles can be removed by the use of *weak salicylic acid* (p 3126). The *mercurial preparations* (p 3121) remove the freckles but are dangerous because their persistent use may lead to deposition of metallic mercury in the tissues with a permanent grayish pigmentation of the skin. Caustic acids and electrodesiccation are inadvisable since they may result in scar formation.

HERPES SOLARIS

Herpes solaris does not differ from *herpes simplex* (p 3289) except that it is regularly evoked in certain individuals by exposure to sunlight. It affects the face and lips.

PHOTOSENSITIVITY

The changes resulting in the skin from exposure to ultraviolet rays already described represent average responses of the normal skin. The material which follows deals with conditions in which the *abnormal reaction* is the result of a *photosensitivity* that is altered capacity to react to normal light stimuli.

Lupus Erythematosus—Lupus erythematosus elsewhere described (p 3395) frequently makes its appearance after exposure to sunlight. The condition once present is made worse by the effects of ultraviolet radiation. In these instances the rays constitute a precipitating or aggravating factor rather than the actual cause of the disease.

Hydroa Aestivale Porphyria—Hydroa aestivale is a rare eruption seen in childhood during the summer months. It is due to inherent sensitivity of the skin to sunlight, believed to result from an abnormal porphyrin metabolism. *Vesicular* and *bullous eruptions* occur on the exposed surfaces, particularly the face and extremities. The condition tends to recur in the spring and summer and may result in depressed scars at the sites of the lesions. Often it is associated with latent porphyria.

Treatment consists of preventive measures and the application of soothing ointment.

Xeroderma Pigmentosum—Xeroderma pigmentosum is a rare *congenital* skin disease activated by light. It appears chiefly on the face and consists of *pigmented macules* in a skin which is dry and scaly. Eventually atrophic areas, patches of depigmentation, keratoses and telangiectases develop. There is a resemblance to the skin of chronic radiodermatitis. The keratoses often undergo *malignant change* into basal or squamous cell carcinoma and death usually occurs before puberty.

Treatment involves avoidance of exposure to light and *early eradication of the precancerous keratoses*.

Tar Melanosis (Riehl's Melanosis)—Tar applied to the skin as a medicament or in certain occupations may enhance the effect of ultraviolet light. Tar ointments may produce a violent *bullous dermatitis* in those portions exposed to sunlight. Prolonged industrial contacts with tar, lubricating oils and coal dust have led to a dirty, irregular, yellowish, brown or even black pigmentation of the exposed skin known as Riehl's melanosis. It has also been said to occur following the use of face powders containing *aniline dyes*. In addition there may be thickening of the skin with comedones, atrophic changes, telangiectases and keratoses resembling a chronic radiodermatitis. While melanosis may occur in covered portions of the body, it is most frequent and certainly most intense in those areas exposed to light.

The photosensitizing properties of tar have been made the basis of a *therapeutic procedure in psoriasis* (p 3420) by combined use with ultraviolet light.

Urticaria Solaris (Photogenica)—Urticaria solaris is a manifestation of physical allergy. It occurs constantly and recurrently in sensitive individuals and differs in no respect from ordinary urticaria (q v).

Pellagra—The eruption of pellagra (p 3237) is usually most intense in areas exposed to light, the face, back of the neck and dorsums of the hands. Light must be considered as only one of the traumas capable of damaging the susceptible skin of *pellagrins*. Other slight forms ury

may be responsible and this accounts for the appearance of eruption on covered skin surfaces

Eczema Solare—In certain light sensitive individuals an eczematoid eruption may occur on exposed surfaces as the face nape of the neck and dorsums of the hands. The eruption consists generally of discrete papules and vesicles mingled with which may be some urticarial wheals.

Cheilitis Due to Lipstick—A number of dyes applied to the skin are able to produce sensitivity to light. These include *eosin*, *acridine*, *fluorescein* and *methylene blue*. Of practical importance is the cheilitis produced by lipstick dyes which in some instances is due to the photosensitizing action of the contained dye. The cheilitis does not appear unless the wearer is exposed to sunlight.

Perfume Dermatitis (Berlock)—Oil of bergamot contained in toilet waters and perfumes produces photosensitivity in some persons. The clinical condition produced by this action has been called "berlock dermatitis" or pigmentary dermatitis from perfume. After application of the oil of bergamot and exposure to ultraviolet light an inflammatory *erythema* may occur which later gives way to a tan or brownish pigmentation that may persist for many months. The outline of the pigmented area is usually linear or pendant like because of the manner in which these cosmetics are ordinarily applied. The most common site is the neck.

Because of the tendency to produce a long lasting pigmentation oil of bergamot and ultraviolet light have been used in the treatment of *vitiligo* (p 3404).

Photosensitivity Induced by Drugs—Photosensitivity may be produced by the injection or ingestion of *gold salts*, *sulfonamides*, *barbiturates*, *arsenicals* and *rose bengal*. The eruption appears only on those surfaces exposed to light and is generally *pruritic* and *maculopapular*. In extreme cases *vesicular* and *bullous outbreaks* are observed.

Patients receiving gold salts or sulfonamides are warned against exposure to sunlight or ultraviolet lamps.

INJURIES DUE TO ROENTGEN RAYS AND RADIUM

Roentgen rays and radium emanations produce a similar effect upon the skin. Their action is upon the cytoplasm of the cells. Therapeutic doses are ordinarily accompanied by little or no cutaneous reaction but therapy may require the production of an *erythema*.

Besides patients under treatment for cutaneous and visceral disorders dermatoses from roentgen rays and the radium emanations are seen in physicians who use the diagnostic and therapeutic media, dentists, technicians and workers in some industries such as the painting of watch dials. The great tragedy of these afflictions is the insidious onset of the changes appearing many years after the exposure. The changes produced are those of an acute and chronic inflammation of the skin often with *malignant degeneration*.

ACUTE RADIODERMATITIS

Acute radiodermatitis occurs within a variable period after exposure to the rays. The usual interval is only a matter of days. The reaction may consist of *erythema* alone, *erythema* and *edema* or *erythema edema ves*

DIFFERENTIAL DIAGNOSIS OF

Localized Pruritus (Itching)

See also Generalized Pruritus p 3170 Pruritus Vulvae p 2594 Pruritus Ani p 3415 Excoriations p 3186

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|--------------------------|--|
| Angioneurotic edema | Local urticarial swellings due to hypersensitivity Note therapeutic response to epinephrine (p 3347) |
| Atopic dermatitis | Infantile eczema of face Usually a hypersensitivity to a digestant such as milk or eggs (p 3342) |
| Bites | Local punctum due to bedbugs chiggers larvae (creeping eruption) fleas mites (rat or grain itch) filaria (onchocercus) lice (pediculosis) acarus (scabies) helminths (uncinaria or schistosomes) Identify pathogen by local examination Search wearing apparel and bedding |
| Chickenpox | Generalized vesicular eruption, usually most marked on trunk A polymorphous unilocular lesion (p 421) |
| Contact dermatitis | Eczema Usually the result of hypersensitivity to external applications notably from clothing cosmetics therapeutic agents or substances employed in industry Poison Ivy |
| Dermatophytosis | Ringworm of the scalp hands feet or groin Identify fungus (p 40) |
| Dermatophytids | Vesicular lesions of hands and feet due to hypersensitivity to fungous infection |
| Dermatitis medicamentosa | Sensitivity to drugs particularly opiates and cathartics |
| Intertrigo | Dermatitis of contactual surfaces particularly in axillae under breasts and in groin (p 3161) |
| Lichen simplex | Thickened leathery skin causing intense itching and secondary excoriation (p 3168) |
| Lichen planus | Violaceous papules with central umbilication Usually on flexural surfaces of wrists fore arms and legs May be mucosal or on genitals (p 3337) |
| Measles | Generalized exanthem with maculopapular eruption followed by desquamation Itching during pre eruptive and post eruptive phases (p 409) |
| Malaria | Red papules and vesicles causing intense itching and burning Usually on trunk Secondary to high atmospheric or body temperatures (p 3171) |
| Neurodermatitis | Intense itching without dermatosis or with the presence of papules and lichenification Usually of neck and extremities More frequent in elderly females (p 3223) |

| | |
|------------------------------|--|
| <i>Neurotic excoriations</i> | Intense itching without local dermatosis except for secondary excoriation. Most frequent in young females (p 3231) |
| <i>Pediculosis</i> | Of scalp body or pubic regions. Note punctum and lymphadenopathy. Identify nits. Look for lice or crabs in clothing and bedding. |
| <i>Prurigo mite</i> | Due to hypersensitivity. Urticarial eruption of extensor surfaces with marked excoriation. Seek offending allergen (p 3343) |
| <i>Psoriasis</i> | Oyster scale papules. Most frequently observed on trunk, elbows and extensor surfaces. May involve scalp, nails and anogenital regions. Tendency to chronicity with recurrences (p 3414) |
| <i>Ringworm</i> | Fungous infections of scalp, body, axillae, groin or feet. Identify fungus (p 3293) |
| <i>Scabies</i> | Infestation by acarus. Note burrows between fingers and in webs, on volar aspects of wrist, axillary folds, nipples, buttocks, scrotum or penis. Identify parasites (p 3181) |
| <i>Seborrhoea</i> | Dry or oily scaling of scalp producing intense itching (p 3430) |
| <i>Urticaria</i> | Multiple wheals as the result of psychogenic or allergic disturbance. Note therapeutic response to epinephrine (p 3345) |

ules and bullae. Later the skin becomes dry and *pruritic*, burning sensations are marked and hairy areas develop *alopecia*.

When the acute reaction subsides there may be only residual *pigmentation* and some scaling which in time disappears completely. In the severer cases chronic radiodermatitis persists.

The acute phase is treated by application of cool *wet dressings* (p 3134) and bland *antipruritic ointments* (p 3136). No more roentgen or radium therapy should be given to these regions at least temporarily.

CHRONIC RADIODERMATITIS

Chronic changes may eventuate from an acute radiodermatitis or they may appear insidiously months or years after exposure to roentgen rays or radium. The latter variety is usually encountered in physicians, dentists and technicians who have exposed themselves frequently to small doses.

In chronic radiodermatitis the skin may show marked *dryness*, *alopecia*, *wrinkling*, *atrophy* areas of *hyperpigmentation* and *depigmentation*, *telangiectases* and wartlike *keratoses*. If the blood supply has been seriously impaired indolent and extremely painful *ulcers* appear. The *keratoses* are particularly dangerous as they often undergo malignant degeneration into *squamous cell carcinoma* (p 3223).

Treatment—Treatment requires abstinence from further exposure to the rays. Soap is to be avoided for this dries and further damages the skin. *Vegetable oils*, *cold cream* and *bland ointments* are useful applications. *Keratoses* must be considered precancerous and extirpated by *electrodesiccation* or *scalpel surgery*. *Ulcers* are very resistant to treatment but great success has been reported with local application of the juice of the leaf of

Aloe vera a subtropical plant (p 3113) *Cod liver oil ointment* has been recommended as well as 2 to 10 per cent *pectin powder* in sterile aqueous solution as a wet dressing covered with oiled silk (p 3134) Whenever the procedure is feasible excellent results are obtained by *surgical excision* of the diseased skin and *transplantation* of healthy skin

PARASITIC INFESTATIONS AND ANIMAL BITES

The integrity of the human skin is often compromised by parasitic infestation and the bites of animals Systemic complications arise from (1) resultant *bacteremia* with the common ubiquitous pathogens (2) *infection* with specific organisms for which the parasite or animal acts as a vector (3) *toxemia* due to the actual introduction of a poison such as the venom of reptiles and spiders (4) interference with physiologic mechanisms such as the *anticoagulant* effect of hirudin secreted by the leech

The material of the present section is not concerned with the systemic consequences of parasitic infestation or animal bites Attention is focused upon the *local cutaneous disturbances* that are produced by the harbored parasites or that result from the bite of the hit and run assailant

The conditions discussed include scabies (the itch), pediculosis capitis (head lice) pediculosis corporis (body lice) pediculosis pubis (crabs) cimicosis (bedbug bite), pulicosis (flea bite) trombidiosis (chiggers) sand flea bites mosquito gnat and fly bites tick bites creeping eruptions, grain itch rat mite itch uncinari dermatitis schistosome dermatitis leech bites reptile and spider bites bee wasp scorpion tarantula and centipede bites mammalian bites (rabbits cats dogs and rats) and human bites

SCABIES (THE ITCH)

Scabies is a contagious disease caused by the *Acarus scabiei* whose female penetrates the skin and deposits her ova In this process a burrow tunnel or gallery is produced in the stratum corneum The mature parasite is found at the apex of the burrow while the ova are strung out toward the surface

Scabies occurs at any age and in either sex Overcrowding and uncleanness are contributing factors Transmission of the parasite occurs through *direct human contact* Bedding materials toilet seats towels and even paper money may be infested with the parasite and indirectly transmit the infestation Humans may also be affected although more rarely by scabies derived from pet birds cats dogs and camels Under these circumstances, the duration of the disease is usually short unless reinfections occur persistently

Clinical Manifestations—*Itching* is of course the initial complaint and is frequently of great intensity The patient suffers most bitterly at night and is comparatively comfortable in the daytime

The characteristic lesion is the *burrow* which is a linear or curvilinear slight elevation usually yellowish brownish or almost black in color Secondly there appear small *papules* and *vesicles* In the uncleanly and in persistent infestations *excoriation* and *pustules* develop *Impetiginization* may occur from secondary infection In infants and children the condition produces large *vesicles* and *bullae* because of the thin texture of the skin

The *distribution* of the eruption is so characteristic as to the

diagnosis The sites involved include the interdigital surfaces and web spaces of the hands the volar aspect of the wrist the anterior axillary fold the areas about the nipples in the females the abdomen the buttocks the scrotum and the penile shaft In neglected individuals a wider involvement is often seen as well as a more marked and aggravated eruption

Characteristically the face and neck are not involved except in nursing infants who acquire the condition from the maternal breast The palms and soles are also free in adult scabies but involved in infants and children where vesicular and bullous lesions are commonly observed

Diagnosis—The diagnosis of scabies requires a high index of suspicion it should be thought of in the presence of *any itching lesion* especially when the itching is *nocturnal* and the eruption involves the *sites of predilection* Careful search will reveal the typical burrows and the parasite

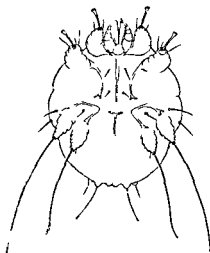


FIG. 899.—Mite causing scabies or itch *Sarcoptes scabiei*

may be demonstrated by the use of a hand lens Ordinarily other members of the family or contacts have similar infestations which require treatment if the patient is to be permanently relieved Such familial or contact infestations are diagnostically corroborative

Treatment—The objectives of treatment in scabies are to destroy the parasites dissipate the secondary eruption and prevent reinfection There was initial hope that treatment might be greatly simplified by the application of DDT in talcum powder or by suspension in an emulsifying agent Unfortunately these hopes have not been sustained by further experience

Previous methods of treatment much more complicated followed the undernoted routine

- 1 *Clothing and bed clothes* are boiled and laundered or preferably sent to a commercial laundry for more complete sterilization

- 2 The patient takes a *warm bath or shower* and vigorously scrubs all portions of the body with *green soap* applied with a fairly *stiff brush* Particular attention is paid to the itching areas The skin is then rubbed dry

3 A *parasiticide* is thoroughly applied to all body surfaces below the neck. Extra attention is given to the itching surfaces which contain the lesions. The preparations in common use include *Sulfur Ointment* USP XII (10 to 15 per cent) (p 3128), *Balsam of Peru Ointment* (5 to 10 per cent) (p 3114), *Benzyl Benzoate* (33 $\frac{1}{3}$ per cent in vanishing cream or made up with equal parts of ethyl alcohol and soft soap) (p 3114) *Rotemone Emulsion* (1 to 2 per cent) (p 3126)

If the sulfur ointment is employed the first application is made in diluted form. Equal parts of the official preparation and cold cream are mixed and tested on the skin. If the diluted preparation does not produce a chemical dermatitis the full strength is employed at the next application.

4 After the application of the parasiticide the patient dons *clean sleeping garments* and retires to a bed that has been previously prepared with *fresh linens*. Clean underwear is laid out for the following morning. The bed, personal clothing and appurtenances are not changed until treatment is completed.

5 The parasiticide is reapplied when the patient awakens in the morning again that night on retiring and for the fourth time on arising the following morning. With the 33 $\frac{1}{3}$ per cent benzyl benzoate or the rotenone emulsion this amount of therapy suffices. In fact it is claimed that one or two applications are enough. The patient then *discards* the night clothes, underwear and bed linen which are boiled in the family laundry or sent out to the commercial laundry. The prolonged bath with scrubbing using green soap and a stiff brush is repeated again paying particular attention to the previously involved areas. The skin is dried and areas of irritation are treated with a simple dusting powder (p 3137) a bland ointment (p 3136) or lotion (p 3137).

When sulfur or balsam ointments are employed it is necessary to continue the treatment for three consecutive nights. The cleansing bath is not taken until the morning of the fourth day.

6 *Reinfestation* is prevented by treating other members of the family or other human contacts similarly afflicted and by scrubbing bedding materials, toilet seats and other objects that possibly harbor the organism. Should reinfestation occur despite the precautions the treatment is repeated and a possible undiscovered source (human or animal) of the infestation is sought.

PHEDICULOSIS CAPITIS

Pediculosis capitis is an infestation of the scalp hair with the head louse. It is attended by *itching*, secondary skin changes and *posterior cervical lymphadenopathy* (p 1136).

Etiology—The head louse is grayish in color and 2 to 4 mm in length. It is seen easily with the naked eye. It deposits its ova or nits and attaches them by a chitinous envelope to the shafts of the hairs a short distance from the scalp. Transmission is usually by direct contact but may be through combs or brushes. Children are most frequently affected and the disease is more common in girls with long hair. It is encountered most often in dispensary practice. This condition is uncommon in Negroes.

Clinical Manifestations—The chief complaint is usually *itching of the head* but this may be absent. The site of predilection is the occipital re-

gion where the hair is thickest. The patient may present an *eczematous* or *impetiginous eruption* in the nuchal region or lower down on the neck with or without lymphadenopathy. Crusted scratched lesions may be present on the face and a blepharitis may occur from rubbing of the eyes.

Diagnosis—The diagnosis of pediculosis is made by seeking and finding the nits. The disease must always be considered in the presence of a nuchal impetigo or eczema with itching or unexplained posterior cervical lymphadenopathy.

Treatment—The introduction of the powerful insecticide DDT (p 3118) has greatly simplified and improved methods of delousing. Two ap



Fig 900—Scabies A Wrists B Genitals



Fig 901—Pediculosis capitis



Fig 902—Pyoderma pediculosis

plications of DDT (p 3118) at weekly intervals provide a clean and satisfactory method of ridding the patient of pediculi. The insecticide mixed with talcum powder or dissolved in kerosene needs only to be rubbed into the parts.

The time honored remedy is equal parts of *kerosene* and *olive oil*. The head is thoroughly shampooed after which the mixture is applied to the entire scalp. The head is wrapped with a towel turban which is allowed to remain in place overnight. In the morning the head is shampooed again, vinegar is rubbed on to soften the nits which are then removed with a fine toothed comb. *Acetic Tincture of Larispur NF* (p 3121) may be used by the same technic employed for the kerosene olive oil mixture.

The use of the proprietary preparation *Cuprex* (Merck) is simpler and highly satisfactory. The *Cuprex* (p 3118) is rubbed into the scalp and hair. Ten minutes later a second application is made and this is permitted to remain untouched for two to four hours. The hair is then fine combed to remove dead parasites and nits. Finally, a cleansing soap and water shampoo is used and the treatment is complete.

The more modern forms of treatment employ the newer chemicals *rotenone* (1 to 2 per cent) *Lauryl Thiocyanate* (25 per cent in Paraffin Oil) and *Lethane* (384 Special) (15 per cent in kerosene). The last named is simple cheap and effectual. The preparation is applied by hand or with

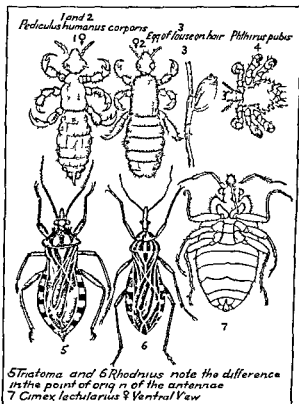


Fig 903—Lice cone-nosed bugs (*Triatoma* and *Rhodnius*) and bedbug (*Cimex*)

a fine spray. The hair is well soaked especially near the roots. The patient keeps the eyes closed during the application of the remedy. Any excess is removed from the ears, neck and forehead. The hair is fine combed as soon as possible and for several days thereafter at frequent intervals. One application kills lice and nits immediately. The hair is not to be washed for several days.

After delousing the secondary skin lesions may be treated with bland ointment (p 3136) or 3 per cent ammoniated mercury if there are evidences of a pyoderma. Reinfestation must be prevented. As a preventive measure combs and brushes are sterilized. Contacts are subjected to the treatment outlined above.

PEDICULOSIS VESTIMENTORUM (CORPORIS)

Pediculosis vestimentorum is an infestation of the clothes with the body louse. The alterations in the skin are produced secondarily by the bites of the parasite with consequent itching and scratching.

Etology—The body louse is somewhat larger than the head louse and grayish in color. When distended with blood it becomes reddish or purple and is easily seen with the naked eye. It is found in the seams of the underclothes but in rare instances may be seen clinging to the body hairs. Ova are deposited in the clothes and have been found on the hairs.

Pediculosis corporis is a disease of filth and overcrowding. It occurs in epidemic form in soldiers and civilians alike when bathing facilities are not available. It is rare in children and more common in men than women. This parasite transmits *epidemic typhus* (p 360), *trench fever* (p 383) and *relapsing fever* (p 357).

Clinical Manifestations—*Itching* is the presenting complaint. The skin reveals secondary changes such as linear scratch marks, excoriations, crusts and pyogenic lesions. The primary lesion is the small red hemorrhagic *punctum* where the bite occurred. In long standing infestation the skin may show considerable *pigmentation* and *thickening* (*Vagabonds Disease*). When neglected serious *pyodermas* may occur especially in persons in poor physical condition.

The *trunk* is almost exclusively affected although at times a few lesions may be present on the arms and thighs. The favored sites are the scapular regions, the buttocks, the belt line of the abdomen and the external aspects of the thighs.

Diagnosis—Finding the louse in the seams of the underclothes establishes the diagnosis.

Treatment—The therapeutic indications are to rid the clothes of the parasites and remedy the secondary skin changes. The outer garments and bed clothes are treated by steam and hot air sterilization. The underwear should be boiled. Before use everything is sprayed with DDT.

The patient bathes and scrubs thoroughly with soap and water. In the morning the pyodermas are treated with 3 to 5 per cent *ammoniated mercury* (p 3121) while a bland ointment (p 3136) or a soothing lotion (p 3137) is applied to irritated areas. The methods of therapy recommended for head lice are also applicable to body lice (p 3183).

PEDICULOSIS PUBIS (CRABS)

Infestation of the pubic hair by the crab louse is attended by itching but by little or no change in the skin.

Etology—The crab louse is smaller than the head or body louse. With its strong claws it attaches itself firmly to the hair and buries its head in the hair follicle. Unlike other lice it is rarely seen moving superficially and it is difficult to dislodge from its position. Ova are deposited upon the hairs and resemble those seen in *pediculosis capitis*.

This condition is often acquired in sexual intercourse but may also be got from toilet seats, beds and other mediate objects. It is seen most commonly in adults and more frequently in males possibly because of greater hairiness.

Clinical Manifestations—The presenting symptom of crabs is *itching* although secondary excoriations and pyodermas may be present. Rounded coin-sized grayish or bluish *macules* which do not disappear on pressure.

The use of the proprietary preparation *Cuprex* (Merck) is simpler and highly satisfactory. The *Cuprex* (p 3118) is rubbed into the scalp and hair. Ten minutes later a second application is made and this is permitted to remain untouched for two to four hours. The hair is then fine combed to remove dead parasites and nits. Finally a cleansing soap and water shampoo is used and the treatment is complete.

The more modern forms of treatment employ the newer chemicals *rotenone* (1 to 2 per cent) *Lauryl Thiocyanate* (25 per cent in Paraffin Oil) and *Lethane* (384 Special) (15 per cent in kerosene). The last named is simple, cheap and effectual. The preparation is applied by hand or with

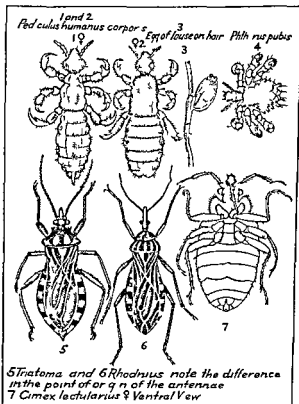


Fig 903—Lice, cone-nosed bugs (*Triatoma* and *Rhodnius*) and bedbug (*Cimex*)

a fine spray. The hair is well soaked, especially near the roots. The patient keeps the eyes closed during the application of the remedy. Any excess is removed from the ears, neck, and forehead. The hair is fine combed as soon as possible and for several days thereafter at frequent intervals. One application kills lice and nits immediately. The hair is not to be washed for several days.

After delousing, the secondary skin lesions may be treated with bland ointment (p 3136) or 3 per cent ammoniated mercury if there are evidences of a pyoderma. Reinfestation must be prevented. As a preventive measure, combs and brushes are sterilized. Contacts are subjected to the treatment outlined above.

| | |
|-----------------------|---|
| Scabies | Note burrows papules vesicles and excoriations particularly of wrists and ankles genitals and abdomen Look for acarus in burrows (p 3180) |
| Seborrheal dermatitis | Dry reddened and scaly macular areas or moist eczematoid plaques accompanying dry or oily seborrhea of the scalp (p 3432) |
| Senile vulvovaginitis | Local atrophy fissuring and leukoplakia. A precancerosis |
| Sycosis vulgaris | Chronic staphylococcal inflammation of hair follicles especially of male beard (p 3249) |

may also be observed These pigmented spots are the *maculae caeruleae* or *taches bleuâtres* and are most frequently observed over the lower back

The disease is usually confined to the *pubic hairs* In hirsute individuals it tends to spread along the abdominal perineal and perianal hairs Infestation of the hair of the thighs and legs abdomen chest axilla and even the eyebrows eyelashes and beard may be found

Diagnosis—Usually the diagnosis of crabs is simple since the *nits* and parasites are readily found at the base of the hairs Difficulty may be encountered in those patients in whom the infestation is numerically slight even though itching is marked If the diagnosis is considered a careful search will usually reveal the nits

The primary condition may be obscured by a treatment dermatitis when the patient first appears The use of strong mercurial ointment may have induced a severe local and even generalized dermatitis

Treatment—The destruction of the parasites and nits and cure of the secondary skin changes are attempted Shaving of the affected hairs is undesirable and frequently leads to skin irritation

The affected parts must be thoroughly washed with soap and water after which the specific remedy is applied (p 3183)

Of these remedies the proprietary Cuprex is to be preferred This substance is rubbed into the affected area The application is repeated in ten minutes and permitted to remain for one or two hours While the hair is still moist a fine comb is used to remove the dead lice and their ova Treatment is completed by a shampoo of soap and water DDT powder in 10 per cent strength also is an effective pediculicide

BEDBUG BITES

The bites of the common bedbug produce an acute eruption characterized by *itching wheals* with a *central hemorrhagic punctum*

Etiology—The bedbug is not truly a parasite of man but does infest beds bedding upholstered furniture walls woodwork and doorways It emerges at night to feed upon man It attacks people of either sex and any age Obviously it is found more often in poor and dirty surroundings The insect itself is 5 to 7 mm in length dark brown in color and whirled emission is a nauseous odor

Clinical Manifestations—*Itching burning sensations* and a *wheel* occur at the site of the insect's bite At first the wheel is typically urticarial

DIFFERENTIAL DIAGNOSIS OF

Excoriations (Scratches)

Excoriations scabs and crusts are seen in all lesions accompanied by itching (p. 3170) sunburn (p. 1951) and conditions formerly grouped as eczemas and now better classified as atopic dermatoses

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|-------------------------------|--|
| Atopic dermatitis | Infantile eczema Erythematous patches papules vesicles and scales Often due to sensitivity to dietestant particularly milk and eggs (p. 3342) |
| Bite | Central hemorrhagic punctum with surrounding wheal and excoriation due to itching May be caused by acarus pediculi bed bugs fleas ticks gnats mites and mosquitoes |
| Contact dermatitis | Hypersensitivity as the result of occupational exposure or applications of therapeutic agents Make patch test (p. 3330) |
| Dermatophytosis | Ringworms caused by fungi Identify pathogen from local scrapings (p. 3293) |
| Dermatophyids | Local vesicular allergic responses due to sensitivity to infecting fungus (p. 3299) |
| Drug eruptions | Variegated dermatoses following oral or parenteral administrations of many substances including barbiturates sulfonamides gold arsenic iodide bromide phenolphthalein aminopyrine atropine salicylate and silver (p. 3335) |
| Exfoliative dermatitis | Generalized scaling Often result of hypersensitivity to chemicals particularly arsenic gold and sulfonamides (p. 3385) |
| Feigned eruptions | Artificially induced lesions as the result of application of chemicals mechanical or thermal trauma (p. 3233) |
| Intertrigo | As the result of constant friction. Identify irritant Correct hygienic errors (p. 3161) |
| Lichen simplex | Thickened violaceous leathery dermatosis accompanied by prolonged itching and excoriation In emotionally unstable patients (p. 3168) |
| Neurotic excoriations | Crustings in regions accessible to the fingers A compulsion neurosis requiring psychiatric therapy (p. 3231) |
| Paget's disease of the breast | A malignant eruption involving nipple or areola associated with carcinoma of the ducts Get biopsy and refer to surgeon for radical mastectomy (p. 2581) |
| Pediculosis | May involve scalp body or pubis Accompanied by local lymphadenopathy Look for nits lice and crabs |
| Prurigo mitis | An atopic dermatitis of childhood characterized by fleshy papules and urticarial wheals Seek offending allergen (p. 3343) |

| | |
|-----------------------|---|
| Scabies | Note burrows papules vesicles and excoriations particularly of wrists and ankles genitals and abdomen Look for acarus in burrows (p 3180) |
| Seborrheal dermatitis | Dry reddened and scaly macular areas or moist eczematoid plaques accompanying dry or oily seborrhea of the scalp (p 3432) |
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BEDBUG BITES

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Etymology—The bedbug is not truly a parasite of man but does infest beds bedding upholstered furniture walls woodwork and draperies It emerges at night to feed upon man It attacks people of either sex and any age Obviously it is found more often in poor and dirty surroundings The insect itself is 5 to 7 mm in length dark brown in color and when crushed emits a nauseous odor

Clinical Manifestations—*Itching burning sensations* and a *wheel* occur at the site of the insect's bite At first the wheel is typically urticarial

Lesions may occur at any site on the body but are most common on the uncolored areas. Thus in pyjama wearers the areas about the ankles, wrists and neck are most affected. A tendency to linear distribution of lesions may also be noted indicating the line along which the insect has crawled in its feeding expedition.

Diagnosis—The diagnosis of bedbug bites rests upon demonstration of the offender in the bed or other articles of furniture. The skin lesion serves merely to arouse the suspicion of practitioner and patient. The sign posts include the distribution of the lesions, the central puncta and the nocturnal exacerbation. Once the diagnosis is considered a search at night will be rewarded by detecting the invader. Few experiences are more humiliating than to make elaborate chemical and other studies relative to an urticaria and see it proved to be the bite of this odious insect.

Treatment—The skin lesions are treated with the utmost simplicity by the application of calamine lotion, boric acid wet dressings or alcohol swabbings.

Most important is the elimination of the pest. This is usually best done by professional "exterminators." Scrubbing of infested wooden furniture with soap and water or 1:500 bichloride of mercury solution, boiling of bedclothing, attention to infestations of walls, wallpaper and upholstered furniture are all important in getting rid of the bedbug.

FLEA BITES

The bite of the common flea produces an itching wheal with a central hemorrhagic punctum.

Etology—The flea (*Pulex irritans*) is ubiquitous and is not truly a parasite of man, using him merely as a source of nourishment. Fleas inhabit the dwelling places of man and thrive on rubbish or lower animals such as dogs, rats and swine. They deposit their ova in their particular habitat and not in the skin of man. Certain fleas are vectors of diseases such as bubonic plague (p. 322). Fleas attack either sex and all age groups. Some individuals seem to possess an acquired immunity to the bites.

Clinical Manifestations—With its bite the flea injects an irritating fluid. The first reaction noted is a *hemorrhagic punctum* surrounded by an area of hyperemia. Itching may be intense. Later a small or large urticarial wheal appears. Scratching induces secondary changes such as excoriations, crusted papules and larger hemorrhagic areas.

Diagnosis—Flea bites produce lesions on exposed parts of the body. If insect bite is considered, no diagnostic difficulty should arise.

Treatment—Simple treatment with calamine lotion, alcohol, boric acid wet dressings or witch hazel is adequate. Pruritus is relieved by the use of 5 per cent camphor or 0.5 to 1 per cent menthol in dusting powder (p. 3137) or lotion (p. 3137). The local application of 3.5 per cent iodine-chloroform or ammonia is often most grateful.

Of greater importance is the necessity of ridding the premises of the insect. The house should be thoroughly cleaned. Breeding sites within and without should be eradicated and infested pets are to be shampooed and dusted with a flea powder.

CHIGGER BITES (TROMBIDIOSIS)

The chigger or *harvest mite* produces bites which cause considerable irritation and discomfort

Etiology—The harvest mite is a larval acarid of almost microscopic size which becomes attached to the skin surface of humans and other animals. It dwells in open fields in underbrush, berry patches and on the leaves of small trees. The chigger is apparently readily able to penetrate through clothing of ordinary thickness.

Clinical Manifestations—The chigger torments humans in the hot months of June, July and August, particularly in the southern and central United States. Their bites are felt most often below the knees, although lesions of the thighs and trunk are not uncommon. They produce a maddening itch and an eruption of firm *papules*, *papulovesicles* or large *vesicles* or *blebs*. *Urticarial lesions* are not infrequent.

Diagnosis—The diagnosis of the chigger bite is dependent upon the history rather than the specificity of the lesions. Seasonal occurrence and exposure to undergrowth in indigenous regions are the most suggestive factors.

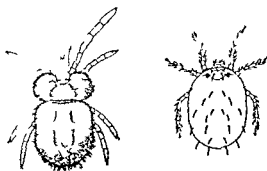


Fig. 903.—The chigger (mite) *Eutrombicula alfreidugesi*. Adult mite left, larva right.

Treatment—The prevention of chigger bites is best accomplished by the use of powdered sulfur mixed with nine parts of *vanishing cream*. This preparation is applied to the body surfaces that are exposed. Dusting the underclothes with *pyrethrum* (p. 3125) or powdered sulfur has been recommended, but the danger of dermatitis due to sensitivity must be borne in mind.

Curative treatment requires a thorough cleansing bath using bland soap and water and the application of a mild antipruritic ointment or lotion (p. 3136) to allay the itching.

SAND FLEA BITE (CHIGOE BITES)

The sand flea (chigoe or *Tunga penetrans*) is able to burrow into the skin and produce ulcerated swellings.

Etiology—The sand flea is a minute insect, about 1 mm in length, which is indigenous to the tropical and subtropical regions of the Americas and Africa. The insect dwells in the soil and the female is more often responsible for serious cutaneous reaction due to its tendency to burrow more deeply.

From Fwings Manual of External Parasites, courtesy of Charles C. Thomas Publishers.

Clinical Manifestations—The insect most often attacks the barefooted person producing lesions between the toes and on the soles. Any portion of the body may be affected. After entry there is marked irritation and itching followed by the formation of an inflammatory nodule. Suppuration, ulceration and lymphangitis are common sequelae. Serious destruction of parts of the foot even amputation in African natives has been attributed to the sand flea.

Diagnosis—There is no difficulty if the condition is considered in regions where the infestation is endemic and especially in those who have walked barefooted.

Treatment—Prevention of chigoe bites calls for wearing shoes in endemic regions. Control of the insects may be effected by keeping the floors of houses clean of soil and other dust and using a little pyrethrum or naphthalene on them.

Treatment of the skin lesions should be prompt. The insect is removed with a sterile needle and the wound dressed with 10 per cent sulfathiazole ointment.

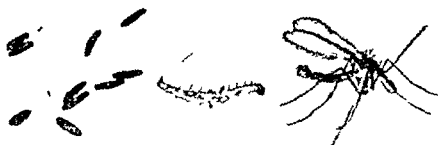


Fig. 906.—Sand fly *Phlebotomus verrucarum*. Eggs (left) and adult male.

MOSQUITO, GNAT AND FLY BITES

Mosquitoes, gnats and flies may produce most irritating bites. These flying pests have added significance since certain varieties of mosquito transmit malaria and yellow fever. African sleeping sickness is transmitted by a fly and even the common house fly may be the carrier of typhoid organisms. These are only a few examples of the important roles of these insects in the transmission of disease.

The bite of the larger flying insect requires no description or consideration from the diagnostic standpoint. Therapy requires the use of a soothing lotion containing an antipruritic (p. 3136).

More important is the use of the insect repellants that are commercially available and the effort to remove breeding places where the insects are known to propagate.

TICK BITES

The bite of the wood tick produces a local inflammatory condition and may introduce into the host the causative agents of severe systemic diseases such as *Rocky Mountain spotted fever*, *tularemia* and *African relapsing fever*.

Etiology—There are a large number of ticks but the most important are those concerned with the transmission of disease to humans and animals. The "wood tick" *Dermacentor andersoni* is widely distributed throughout the northwestern United States and is the chief vector of *Rocky Mountain spotted fever* (p 376). It is also an important agent of

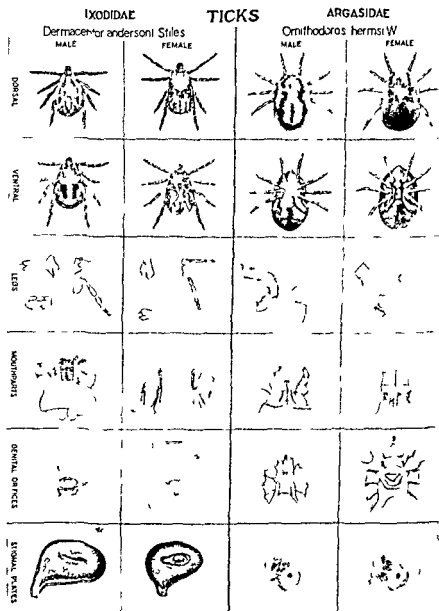


Fig 907—Showing structural details used in classification of ticks. Left Ixodidae (hard bodied ticks). Right Argasidae (soft bodied ticks).

transmission of *tularemia* (p 323). *Texas cattle fever* is borne by a tick *Boophilus annulatus* and *African relapsing fever* by a tick *Ornithodoros moubata*. *Endemic relapsing fever* (p 357) of the United States encountered principally in the southwest is carried by the tick *Ornithodoros tunicata* and *O. hermsi*. Ticks are also vectors of *relapsing fever* in Central and South America.

Hermes Medical Entomology courtesy of Macmillan Company

Tick paralysis is a peculiar form of acute ascending paralysis occurring almost exclusively in children. It is due to a tick bite on the nape of the neck or along the spine and removal of the tick is usually followed by complete recovery.

Clinical Manifestations—The tick buries its beak in the human skin and then swells considerably after sucking blood. It may produce an appearance suggestive of a small tumor. If allowed to remain for a period of time considerable local inflammation may ensue.

Diagnosis—The diagnosis is obvious on close inspection and identification of the parasite in the skin.

Treatment—*Prevention* necessitates the avoidance of tick infested areas, the use of protective clothing and inspection of the body and early removal of ticks. The proofing of homes in endemic areas from the insects and infested small rodents is advisable. Dipping of cattle and exclusion of them from tick infested regions has proved very successful.

Care must be taken to extract the insect in toto. If a portion remains it may induce persistent irritation and inflammatory change. The grasp of



Fig. 903.—Creeping eruption. Lesions on the wrist. Courtesy of Drs. J. B. and Bedford (Science).

the insect may be relaxed by the application of a lighted cigarette, a drop of spirits of turpentine, or a spray of ethyl chloride. Glycerin, kerosene, or mineral oil applied for an hour or more plugs the respiratory orifices; the insect dies and drops off or may be brushed away. Should the tick or a part of it become encapsulated and form a nodule, it must be removed surgically. The insect should never be torn away brusquely, as the head may become detached from the body and remain buried in the skin.

CREEPING ERUPTION (DERMAL CREEPING MYIASIS)

The creeping eruption is a condition in which a parasite burrows in the skin and produces a raised, reddish linear eruption which usually assumes a bizarre pattern.

Etiology—The disease is seen fairly frequently in the southern United States, especially in Florida. Cases have been reported as far north as New Jersey. Most infestations seem to occur on the sands of bathing beaches. No age or sex is immune to this disorder.

The commonest parasite is the larva of the bot fly *Gastrophilus haemorrhoidalis*. This larva burrows into the epidermis lying in the lower portion of the stratum corneum or

upper portion of the rete mucosum. Here it develops into the parasite which tunnels through the epidermis, a noticeable increase in extent being visible daily.

Traumatic dermal myiasis refers to the infestation of wounds by fly larvae. It usually occurs in hot climates and produces an inflammatory, suppurating wound.

Clinical Manifestations—The lesion is strikingly characteristic in appearance. It consists of a single reddish *linear elevation* which may be many inches in length. It is usually curved and gyrate in outline, but never does the line cross itself at any point. Multiple infestations may be present.

No area is exempt from involvement, but most often the lesions are seen on the buttocks, thighs or legs. It may occur on the face and even



Fig. 909.—Grain itch. One of a number of patients with grain itch appearing as an outbreak in an institution from the use of new straw mattresses. (Courtesy of Dr. Joseph V. Klauder).*

involve the mucous membranes. The sole subjective symptom is moderate itching, although this may be absent.

Diagnosis—The characteristic eruption is difficult to mistake. At times when much itching is present, secondary excoriations and pyodermas obscure the pattern and produce a puzzling picture.

Treatment—Various forms of therapy have been used with success. These include (1) painting the area with *Tincture of Iodine*, (2) freezing with *ethyl chloride*, (3) freezing with *carbon dioxide snow*, (4) removal of the parasite with a *needle*, (5) painting the area with 2 to 4 per cent suspension of *salicylic acid in collodion*.

* Andrews, *Diseases of the Skin*.

Traumatic dermal myiasis may be treated by destroying the larvae by irrigating with a solution of 15 per cent *chloroform* in a light vegetable oil. All the larvae must be destroyed after which 10 per cent *sulfathiazole* is applied.

GRAIN ITCH

Grain itch is a generalized itching eruption due to the grain mite (*Pediculoides ventricosus*) acquired from contact with infested straw or grain. The affliction is most common from May to September. The lesion is characterized by *urticarial wheals* with central tiny *vesicles* or *pustules*. As in scabies the itching is most intense at night but the type of lesion and its wider distribution aid in differentiation. It is distinguished from urticaria by the unusual central vesicle or pustule and the fact that the lesion persists instead of being evanescent. The mite does not burrow into the skin so that it can only be found in the infested grain or straw (mattress).

Treatment requires removal from the source of infestation, disinfection of the clothing and straw mattresses by fumigation and the application of soothing lotions (p 3137) or ointments (p 3136).

RAT MITE DERMATITIS

Rat mite dermatitis is an itching eruption consisting of urticarial wheals and papulovesicles with central hemorrhagic puncta caused by bites of a mite whose chief host is the common brown rat. It is of importance since this insect may transmit the organism of *endemic typhus fever* (p 375).

The mites must be destroyed by the eradication of rats. The eruption is treated with soothing lotions (p 3137).

UNCINARIAL DERMATITIS

Uncinarial dermatitis is an itching eruption of the soles and toes characterized by papules, vesicles and bullae. This outbreak is common in the southern United States in those who walk *barefoot*. It is produced by the penetration into the skin of the ova of the *hook worm* (p 1904). The eruption may readily be mistaken for that of dermatophytosis. From the skin the ova enter the blood stream and finally lodge in the intestinal tract where they induce the syndrome of *hookworm disease* (p 1903).

The skin lesions are of minor importance and may be treated with antiseptic solutions (*boric acid*) and mild mercurial salves (*ammoniated mercury ointment*).

SCHISTOSOME DERMATITIS

Schistosome dermatitis is an itching eruption of the body characterized by wheals and later the appearance of *papular* and *pustular lesions*. It is caused by the penetration of a schistosome present in fresh water, the schistosomes being parasites of fresh water snails. The condition is endemic in the northern section of the Middle Western United States and is usually observed after swimming.

Soothing lotions (p 3137) may be applied.

THE LEECH BITE

The cervical glands of the common leech (*Hirudo*) secrete *hirudin* which delays or hinders the coagulation of blood inside and outside of the body

Leech bites are encountered as accidental disturbances in those who wade or swim in infested waters. Secretion from the bite may lead to an annoying local irritation and should be removed. This is readily accomplished by the application of a strong salt solution.

The therapeutic use of leeches was once common practice as a method of counterirritation. There is no justification for its continued use although it is still employed in primitive types of practice.



Fig 910—Schistosome dermatitis—papular eruption on leg

THE BITES OF REPTILES AND SPIDERS

In the United States venom may be introduced by *vipers* (the water moccasin, copperhead, rattlesnakes and the harlequin snake of the south eastern United States) and the *spider* known as the Black Widow, shoe button, hourglass or T spider (*Latrodectus mactans*).

Clinical Manifestations—The clinical manifestations of venomous snake or spider bites are quite similar. Locally there is an intense *cellulitis* at the site of the bite. This is usually on the hands or feet in the case of snakes and often in the genital region with Black Widow spider bites since this particularly vicious assailant often dwells in outdoor privies.

Snake Bite—The clinical manifestations of snake venom are produced by (1) the hemolysis of the red blood cells or (2) the paralytic action on nervous tissue. Because of the former there is a tendency to *extravasation* and *thrombosis*. The depressant action on the nervous system gives

rise after a period of approximately four hours of incubation to *drowsiness* and *ascending paralysis* of the spinal cord reaching the vital medullary centers with *respiratory failure* and *asphyxial convulsions* before death.

Spider Bite—The bite of the Black Widow spider is followed in fifteen to thirty minutes by excruciating generalized pain. There may be *muscle spasm* and board like rigidity of the *abdominal wall*. The patient becomes *restless* and anxious and signs of *shock* appear. The abdominal syndrome of pain and muscular rigidity may be so confusing especially when the spider bite is unnoticed as to lead to unnecessary surgery.

Treatment Snake Bite—The treatment of snake bite consists in the *immediate application of a tourniquet to the limb above the site of the bite*. The wound is then enlarged with crucial incisions 1 inch or more across and venom is sucked out by means of a special cup or orally if there is no other means available. The tourniquet is released for a few seconds every ten or fifteen minutes in order to prevent gangrene below the level of the compression.

As soon as is possible *antivenin* which is commercially available is injected locally around the site of the bite. The remainder is given intramuscularly. The patient should be reassured since the majority of snakes are not venomous and a fatality occurs in only perhaps 10 per cent of those bitten by known venomous snakes. The official polyvalent snake venom of North America is Antivenin (Mearctic Protalidae) (p 83). This preparation is effective against *rattlesnakes*, *water moccasins* and *copper heads*.

The bitten patient is removed to an institution as soon as possible and an *intravenous drip* is instituted with 5 or 10 per cent glucose in saline solution. *Blood transfusion* is arranged if there is considerable hemolysis. Sedative drugs such as *alcohol*, *opiates* and the *barbiturates* are avoided since they may add to the respiratory depression resulting from the venom itself.

Spider Bite—Patients with spider bites should also be hospitalized. They obtain the best symptomatic relief from prolonged *hot baths*, *neostigmine*, *opiates* and the intravenous administration of 10 cc of 10 per cent *calcium gluconate*. An antivenin also is available.

THE BITES OF BEES WASPS SCORPIONS TARANTULAS AND CENTIPEDES

There is no evidence that a significant systemic disturbance accompanies the bites of bees, wasps, scorpions, tarantulas or centipedes though popular belief is to the contrary with regard to the latter three. Of course severe shock and even death has been observed from bee stings in persons who manifest hypersensitivity to bee venom.

Locally the bee and wasp produce an intense burning and pain with the production of swelling and edema. Only local treatment is required by the use of cold applications. Bee venom has been recommended in the treatment of *arthritis*. There is no evidence to show that this measure has any particular value.

THE BITES OF MAMMALIANS (RABBITS CATS DOGS AND RATS)

Mammalian bites are of importance since they may be the mechanism by which serious and at times fatal diseases are introduced into the hu-

man body. The rabbit is a carrier of *tularemia*. Cats and dogs harbor *rabies* (p 439). The rat transmits *rat bite fever* (p 363).

The *local treatment* of the bite is mainly concerned with cauterization with fuming nitric acid. This drastic treatment has the recommendation of custom, but recent investigations suggest that *cleansing with soap and water* and *irrigation with saline solution* produce as much benefit with considerably less trauma. The practitioner who is confronted with a *mam malrin* bite will protect himself and his patient if he continues to use cauterization except on the face. Thereafter *rabies vaccine* (p 78) should be given when indicated.

HUMAN BITES

Human bites are important since they may lead to serious *anaerobic infection* of the cellular tissues and also on occasions may be the portal of infection for *syphilis*. It is the practice of the Police Department of New York City to send officers suffering from human bites for *dark field examinations* for the spirochete of *syphilis*. Routine serologic tests of the blood are performed for a period of at least three months thereafter and the patient is not discharged until all possibility of syphilitic infection is eliminated (p 336).

When *anaerobic infection* occurs it is due to *fusospirochetes* (p 355). These infections are usually serious and involve the loose cellular structures. They are seen most commonly on the hand and here may involve the tendon sheaths and produce permanent functional impairment (Fig 1164 p 3974).

Treatment is surgical. Wide incision and drainage of the infected tissues with *irrigations* and *dressings* of warm hydrogen peroxide (1 part hydrogen peroxide to 3 parts distilled water) or warm potassium permanganate solution (1:3000) are indicated. Systemic penicillin therapy is recommended as a measure of preventive medicine.

CHAPTER 14

THE CUTANEOUS SYSTEM NEOPLASMS NEUROGENIC AUTONOMIC, PSYCHOGENIC AND VASCULAR DISTURBANCES

Benign Neoplasms of the Skin

Angioma
Lymphangioma
Moles or Nevi
Senile Sebaceous Adenoma
Fibroma
Lipoma
Multiple Lipomatosis
Neuroma
Multiple Neurofibromatosis
Myoma
Paraffinoma
Glomus Tumor
Stratocystoma Multiflex
Sebaceous Cyst
Synovial Cyst
Multiple Benign Cystic Epithelioma
Steirocystoma

Precancerous Dermatoses

Benign Neoplasms Which Occasionally Assume Properties of Invasiveness

Malignant Arising in Dermatosis Caused by Chronic Irritation or Infection

Malignant Change in Lesions Associated with Gonadal Decrease

Malignant Neoplasms

Basal Cell Epithelioma (Rodent Ulcer)

Squamous Cell Epithelioma

Ingestive Disease of the Breast

Bowen's Disease

Melanocarcinoma

Sarcoma

Pathologic Multiple Hemorrhagic Sarcoma

Metastatic Carcinoma

Lymphosarcoma

Neurogenic Autonomic and Psychogenic Disturbances of the Skin

Neuritic Atrophy of the Skin

Perforating Ulcers

Malignant Erythema

Causalgia

Localized Neurodermatitis

Erythema

Erythromelalgia

Progressive Facial Hematophagy

Hereditary Lymphedema (p. 310)

Neurotic Excoriations

Feigning Eruptions

Tritobromism

Dermatophytosis

Urticaria (p. 3345)

Angioneurotic Edema (p. 3349)

Vascular Lesions of the Skin

NEOPLASMS OF THE SKIN

New growths of the skin may be benign or malignant. Certain of the benign skin lesions display a tendency to undergo malignant degeneration. These are termed precancerous and their recognition is of vital importance since they are most successfully eradicated before they assume the characteristics of invasiveness. Malignant tumors arise primarily in the skin though there may be metastatic deposits from a lesion elsewhere in the body. Direct invasion of the skin by cancer is particularly frequent with untreated malignancies of the female breast.

BENIGN NEOPLASMS

Benign neoplasms are excessive developments of normal tissue. They grow in an orderly fashion and lack the invasiveness and destructiveness of the malignant tumor.

ANGIOMA

Angiomas are vascular nevi produced by dilatation and proliferation of the small vessels of the corium. In exceptionally large lesions the vessels



Fig 911 —Capillary nevus (strawberry mark)



Fig 912 —Hemolympangioma



Fig 913 —Hemangioma



Fig 914 —Port wine stain



Fig 915 —Nonpigmented mole



Fig 916 —Pigmented mole

of the subcutaneous tissue are also involved. While angiomas are congenital abnormalities, usually obvious at birth, the senile type may not become visible for many years.

Strawberry Mark (Simple Angioma) —The strawberry mark is a simple

angioma that appears as a flat slightly raised red or purplish area which may vary from a fraction of an inch to about 2 inches in diameter. The outline is round or irregular and is sharply set off from the normal skin.



Fig 917—Hairy mole



Fig 918—Multiple neurofibromatosis (on Recklinghausen)



Fig 919—Acneiform eruption due to Helio wax

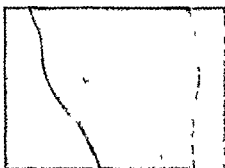


Fig 920—Glomus tumor



Fig 921—Leukoplakia



Fig 922—Senile keratosis

An especially common variety of simple angioma consists of a circumscribed nonelevated dull red area found over the *nuchal region* in almost 50 per cent of infants. Since this abnormality is connected to the vascular tree it becomes engorged and purplish when the infant cries or

struggles. The strawberry mark is also seen on the face and neck. It may be present on any portion of the extremities or trunk (Fig 911)

The progression of a simple angioma is inappreciable at first. Occasionally spontaneous closure of the vessels occurs with the production of an atrophic scar. This type of spontaneous cure may follow an inflammatory reaction due to trauma or pyogenic infection.

The strawberry mark requires no treatment except for cosmetic reasons. Under those circumstances it is wiser for the practitioner to refer the patient to the specialist who may obliterate it by the application of carbon dioxide snow or radium. Other therapeutic procedures are applications of acids, injections of sclerosing solutions and electrodesiccation.

Cavernous Angioma—The cavernous angioma is larger and more deeply situated than the strawberry mark. It appears as a circumscribed well demarcated tumor which may be diffuse or taper off into the normal structures. The color may vary from a vivid red to dark blue. More deeply situated tumors do not affect the color of the overlying skin and may be mistaken for nonvascular neoplasms or cysts.

Cavernous angiomas appear most commonly on the head and neck, although other areas of the body may be affected. The lesion may be as small as a grape or as large as a melon. In the larger tumors other tissues such as muscle, bone and cartilage are often involved.

The cavernous angioma is obliterated by the injection of a sclerosing solution such as sodium morrhuate (p 3941). From 0.2 to 10 cc is injected into the lesion and a satisfactory result may be obtained by mere introduction into the soft tissues rather than directly into the lumen of a vessel. With larger tumors more than one injection may be required. Because of the importance of a good cosmetic result it is often the better part of wisdom to refer such patients to the specialist. In many instances the best result is obtained by the application of radium. In smaller lesions electrodesiccation may be used successfully.

The Port wine Stain (Nevus Flammeus)—The port wine stain appears on the face as a flat purplish or bluish red unilateral lesion of variable size. It may involve only a small portion of the skin or it may spread over the entire half of the face and extend onto the scalp, ear and neck. Extensive stains spread into the mouth and involve the buccal mucosa. Particularly large facial nevi may also involve the orbital vessels producing some degree of exophthalmos. Port wine stains have also been observed although less frequently on the neck, forearm and hand (Fig 914).

The treatment of the port wine stain is most unsatisfactory and should not be attempted by the practitioner. The expert may attempt removal of the smaller nodular tumors by electrodesiccation or the application of radium. It is safer and more satisfactory however to persuade the patient to endure the deformity and conceal the lesion in so far as possible by the use of a commercially available preparation such as Covermark.

Senile Angioma—The senile angioma is very frequently observed on the trunk of elderly persons. The lesion may be of variable size and appears as a raised soft bright red tumor. Multiple lesions usually exist. Consequently there is little to be gained except cosmetic improvement by

removing isolated lesions. These lesions are not truly angiomas of congenital origin but rather localized dilatations of capillaries.

If the patient demands treatment the vessels may be effectively destroyed by *electrodesiccation* (p. 8702).

Anemic Nevus—The anemic nevus is a rarity. It is probably due to a congenital absence or deficiency of blood vessels rarely to local vascular neurosis with vessel spasm. The lesion appears as one or several circumscribed white *macules* which may appear on the face or trunk. These are irregularly rounded and vary from a fraction of an inch to about 1 inch in diameter.

Local therapy is of no avail.

Telangiectasis—Most telangiectases are acquired dilatations of the blood vessels of the skin. They appear as fine red lines in the skin from which the blood can be expressed by pressure. Telangiectases are seen in association with many cutaneous diseases among which are *rosacea*, *basal cell epithelioma*, *chronic radiodermatitis*, *lupus erythematosus*, *xeroderma pigmentosum* and *chronic actinic dermatitis*.

Aside from the isolated telangiectases and those associated with other cutaneous disturbances the vascular anomaly may assume the appearance of the spider nevus or it may exist as a multiple systemic disturbance. The *spider nevus* (*nevus araneus*) occurs principally upon the face and consists of multiple dilated capillaries radiating from a central red slightly elevated pinhead sized vessel.

Multiple hemorrhagic familial telangiectasis (Rendu Osler Weber's disease) is a rare disease which is hereditary and familial. The first manifestations of the disease usually appear in infancy or childhood. The most frequent symptom is *recurrent epistaxis* (p. 2123) although there may be hemorrhages from other orifices such as the mouth, lungs, stomach and rectum. Besides the multiple telangiectases of the skin dilated vessels of the vermilion surface of the lips, the mouth and the nasal mucosa are especially notable. The telangiectases of the skin may be trifling or so extensive as to appear generalized. As a result of hemorrhage there may be a *secondary anemia* but there are no other changes in the blood. The condition can result in chronic invalidism from severe anemia. Death from hemorrhage has been reported.

Treatment—Telangiectases are usually treated by *electrolysis* with the galvanic current. In the spider nevus the needle is inserted into the central vessel and 1 to 2 ma. of current allowed to flow for five to fifteen seconds or until the area blanches. In *rosacea* and other diseases where the telangiectases may need separate therapy electrolysis is also applied.

Multiple hemorrhagic familial telangiectasis requires systemic therapy. The most satisfactory control of bleeding has been achieved by periodic injections of *moccasin snake venom* (1:3000). Accessible telangiectases of the mucosa can be destroyed by *electrolysis*.

LYMPHANGIOMA

Lymphangiomas are rare congenital abnormalities composed of dilated and proliferated lymphatic vessels.

Simple Lymphangioma—The simple lymphangioma appears as a rounded

raised *tumor* situated in any part of the body. The nature of the lesion is suspected when upon puncture *clear lymph* exudes. Involvements of the tongue and lips give rise to *macroglossia* (p 1685) and *macrocheilia* (p 1683) respectively.

Circumscribed or Cavernous Lymphangioma—The circumscribed or cavernous lymphangioma is a rare lesion occurring usually in the *axilla* where it appears as an irregular grouping of small slightly elevated non-inflammatory vesicles. Upon puncture there is a discharge of colorless lymph.

Hemolymphangioma—Hemolymphangiomas are admixtures of lymphatic and angiomatous elements which have features of each of the more fundamental abnormalities.

The *treatment* of the various types of lymphangioma is best conducted by the *specialist*. For the larger lesion excision is desirable. Favorable results have also been obtained by the injection of sclerosing solutions and the application of roentgen rays or radium.

MOLES OR NEVI

A variety of *fleshy tumors* involving the nonvascular structures of the skin occur as congenital abnormalities and may be present at birth or may appear at some later date even in adult life. Many of these seem to be stimulated to growth at the time of *puberty* when they enlarge rather rapidly. In some instances *pregnancy* seems to be an activating influence.

For the largest part, the moles and nevi have no important significance although they may require removal for cosmetic reasons. In rare instances they are *precancerous* hence their removal is mandatory when they show manifestations of *growth* or *ulceration* (Figs 911 to 916).

The Nonpigmented Mole—The nonpigmented mole is a raised soft skin colored lesion usually about the size of a pea. If removal is required they are best excised for histologic examination although a good cosmetic result may be obtained by *electrodesiccation*.

The Pigmented Mole (Nevus Pigmentosus)—The pigmented mole is an elevated pale tan to deep brown or black lesion varying in size from a fraction of an inch to several inches. It is usually soft or moderately firm rounded in outline and appears most commonly on the *face*, *neck* and *trunk*. Pigmented moles are more commonly multiple than single.

Treatment—The slate colored or bluish black hairless or smooth mole has a potentiality for degeneration into *melanocarcinoma* (p 3225) hence early and wide *excision* is highly desirable whenever inflammation, ulceration or sudden growth occurs. *They should never be treated by intermittent electrodesiccation*.

When a blue black mole is located in an area where it may be irritated as on the toe or finger or where the belt or suspender rubs it is best to remove it by complete *scalpel excision* including an adequate border of healthy skin and extending down to the fascia or muscle. If histologic study of the tissue reveals the presence of malignant change the regional lymph nodes should be dissected out completely and roentgen therapy given postoperatively.

Hairy Mole (Nevus Pilosus)—The hairy mole is a soft raised neoplasm that is skin colored or pigmented and similar in every respect to a pig.

mented mole except that it contains a growth of hair. The hairs may be few or many in number they may be downy or stiff short or long.

The hairy mole rarely becomes malignant but may require removal for cosmetic reasons. The hairs may be epilated by *electrolysis* and the mole may then be destroyed by *electrodesiccation* or removed by *scalpel dissection*.

Cerebriform Mole—The cerebriform mole consists of a grouped lobulated firm tumor of considerable size. It is most often observed on the *scalp* and *neck*. Its importance lies in the fact that malignant degeneration may ensue if the lesion is not completely removed.

Because of the necessity for histologic examination the cerebriform nevus should be *excised*.

Giant Mole—The giant mole is an extensive pigmented hairy nevus that occurs on the *trunk* or *extremities*. It may occupy more than half of the surface area and within the mole there may be many smaller verrucous masses.

The extent of the giant nevus precludes any possibility of excision or removal. Fortunately malignant degeneration occurs rarely.

Linear Mole—The linear mole is a unilateral whip like mass of closely crowded verrucous substance of a pale yellow to deep brown color. Its distribution often follows the pathway of a nerve but it has no connection with any of the underlying neurogenic tissues.

The smaller linear moles may be removed by *excision* or *electrodesiccation*. The larger lesions present a surgical problem which may be solved by extensive plastic procedures.

Fatty and Sebaceous Moles (Nevus Lipomatodes and Sebaceus)—Uncommonly moles contain fatty tissues sebaceous glands sweat glands and occasionally nerve elements. None of these features is usually apparent until histologic examination has been performed.

Blue Nevus—The blue nevus is a rare lesion most often seen on the *face* and *hands* where it appears as a rounded or oval slightly elevated blue papule of variable size. It bears no relation to the *mongolian spot* which appears over the lower back and sacral regions in the darker races particularly the Orientals. The blue nevus and mongolian spot are characterized by the unique fact that they contain pigment producing cells in the *cutis*.

The blue nevus rarely but occasionally degenerates into *melanosarcoma*. Hence it should be *excised* wherever feasible and in all cases where there are manifestations of growth or ulceration.

SENILE SEBACEOUS ADENOMA

The senile sebaceous adenoma appears as one or several lesions in the *middle portion of the forehead of elderly persons*. Each of the neoplasms appears as a pea sized flat papule faintly yellowish and often umbilicated.

Since these lesions in rare instances have been known to degenerate into basal cell epitheliomas they should be examined periodically for evidence of growth. Or they may be eradicated by the application of *carbon dioxide snow* or light *electrodesiccation*. See also *Adenoma Sebaceum* (p. 3148).

FIBROMA

The fibroma appears as a hard or soft growth of variable size usually composed of fibrous tissue enclosing vascular and cellular elements. The soft fibroma is a rounded sessile mass skin colored and symptomless. The hard variety is firm rounded or flat yellowish or brownish and is most commonly located upon the legs.

Fibromas have no importance except in the rare instances where sarcomatous degeneration has been reported. *Excision* under local anesthesia is the recommended method of therapy.

LIPOMA

The lipoma is a tumor composed of adipose tissue. Lipomas may occur as single or multiple growths arising in the subcutaneous fatty layer. They are most commonly observed on the trunk, nuchal region and the extremities but they may occur in other situations. The individual tumor may vary from the size of a filbert to a mass larger than an orange. It is usually rounded, elevated, soft and movable over the deep structures. The tumor is characteristically *lobulated*; this structure often being brought out clearly by pressure. The superjacent skin is normal in appearance.

In simple lipoma the only effective treatment is *excision*.

MULTIPLE LIPOMATOSIS (ADIPOSIS DOLOROSA)

Adiposis dolorosa or Dercum's disease is characterized by the appearance of *painful fat masses* symmetrically arranged. The condition is undoubtedly due to some unknown disorder of metabolism probably related to the endocrinopathies.

Unfortunately, neither local nor systemic therapy is of any avail. The particularly painful nodules may be treated by methods of *injection* (p. 3941) for symptomatic relief.

NEUROMA

The neuroma is a rare growth composed of fibroelastic tissues and nerve fibers. In the few recorded examples the patients were adult male with multiple *painful and tender tumors* of variable size. The lesions were located on the arms, thighs or buttocks and were pink to purple in color. Treatment consists of *excision*.

MULTIPLE NEUROFIBROMATOSIS (VON RECKLINHAUSEN'S DISEASE)

Multiple neurofibromatosis is a *congenital disorder* which has a definite but not constant familial tendency. It is believed to be due to some disturbance of the germ plasma. The condition occurs in both sexes.

Clinical Manifestations—The lesions which consist of *fibromas* and *pigmentations* usually appear in childhood and become more prominent at puberty. There may be only one or two fibromas or hundreds. They vary in size from a pea to a melon, the larger tumors weighing several pounds. They are rounded, soft, skin colored or bluish and often may be invaginated completely into the skin by the finger. They occur with greatest profusion on the trunk but they may be seen on the extremities and even in the mouth. Tumors may be present in the lower bowel and there may be cyst like alterations in the bones especially of the pelvis.

tumor may become stretched and hang over the trunk (*fibroma pendulum*) like a veil (Fig 918)

The pigmentations may be multiple discrete tan freckle like lesions or larger irregular patches of distinctive café au lait color The pigmented areas are not elevated The so-called *forme fruste* variety of this disease displays only the characteristic pigmentation tumors being entirely absent There is no successful therapy *Excision of the tumors is not advised* since this may precipitate sarcomatous change

MYOMA (LEIOMYOMA)

A myoma or leiomyoma is a rare skin tumor composed of smooth muscle The lesion arises from the unstriated muscle of the *arrectores pilorum* or the blood vessels It is most frequent in adults The site of predilection is the anterior surface of the legs but nodules may also occur on the trunk and face Generally the lesions are multiple raised yellow to brown and movable with the skin They are usually tender and at times intensely painful

Treatment is not entirely satisfactory but successful results have been obtained with excision electrodestruction and freezing with carbon dioxide snow

PARAFFINOMA

A paraffinoma is a *foreign body tumor* produced by injection for cosmetic purposes of paraffin or some other oily substance into the skin Reputable physicians do not use this procedure but it has been practiced by enough charlatans so that the dreadful results are still visible today It was and possibly still is used to remove wrinkles and remedy other facial defects From six to fifteen months after the injection firm reddened masses appear in the skin These involve the entire cutis and may be of large size (Fig 919)

Successful therapy requires the *surgical removal of the paraffin* after which roentgen rays or radium may effect a satisfactory cosmetic result

GLOMUS TUMOR

A glomus tumor is a rare growth composed of nerve fibers smooth muscle and blood vessels It is located most frequently in the *terminal phalanx of the digits beneath the nail bed*

The lesion consists of a pea sized or smaller rounded tumor which may produce slight elevation of the skin The most distinctive and diagnostic feature is the extraordinary and excruciating pain produced when the area is pressed or squeezed

Cure is effected by *excision*

STEATOCYSTOMA MULTILEX

Steatocystoma multiplex is a rare condition in which hundreds of small *sebaceous cysts* appear on the body especially the anterior surface of the trunk less often the back arms and thighs The eruption consists of many smooth skin colored variously sized rounded elevations attached to the skin externally They are generally unassociated with acne or seborrhea and are easily removed by a small incision and expression of the cheesy or oily contents

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vessels The lesions increase gradually to a maximum size after which they remain static

The history, multiplicity and character of the lesions usually aid in differentiating this condition from basal cell epithelioma though in rare instances these lesions have undergone malignant transformation

Treatment requires removal with minimal cosmetic defect by *curettage* or light *electrodencation*. At times *scalpel excision* is preferable for larger lesions. *Roentgen therapy* has been successful

SYRINGOCYSTOMA

Syringocystoma is a rare condition consisting of *multiple* small tumors composed of *cysts* lined with epithelium similar to that of the sweat glands. It occurs almost wholly in *young adult females* the lesions being distributed mainly over the *anterior surface* of the trunk. The tumors may be present also on the neck, shoulder, extremities and face

The individual *lesion* is a pea sized or smaller slightly elevated yellowish to brown smooth nodule which in extremely rare instances becomes converted into a basal cell epithelioma

Treatment is not particularly successful but local extirpation has been effected by *electrodencation*, *excision* or *carbon dioxide snow*. A few successes have been claimed for treatment with *roentgen therapy* but the improvement is said to be temporary

THE PRECANCEROUS DERMATOSES

The potentiality for malignancy is occasionally assumed by skin conditions which in most other instances are essentially benign. The recognition of these precancerous dermatoses is of the greatest practical significance as a prophylactic approach to the problem of cutaneous cancer

In a broad and general way the precancerous dermatoses include (1) *usually benign neoplasms*, (2) *dermatoses resulting from chronic irritation and infection*, (3) *cutaneous manifestations of diminution or cessation of gonadal activity in male and female* and (4) a *miscellaneous group*

BENIGN NEOPLASMS WHICH OCCASIONALLY ASSUME THE PROPERTIES OF INVASIVENESS

The benign neoplasms which occasionally become malignant and assume the powers of invasiveness have been previously described (p. 3109). They include

Pigmented Moles (p. 3204)

Blue Nevus (p. 3205)

Senile Sebaceous Adenomas (p. 3205)

Fibromas (p. 3206)

Multiple Neurofibromatosis (von Recklinghausen's Disease) (p. 3206)

Multiple Benign Cystic Epitheliomas (p. 3208)

Cerebroid Mole (p. 320)

Sebaceous Cyst (p. 3208)

Syringocystoma (p. 3209)

The treatment of the precancerous benign neoplasms rests on their clinical appearance and the feasibility of eradication. A single growth such as a mole, adenoma or fibroma is best excised for histologic examination

SEBACEOUS CYST (WEN)

A sebaceous cyst is an enlarged sebaceous gland containing *sebum* and *epithelial debris*. Sebaceous cysts result from closure of the excretory duct. Within the gland secretion continues but retention produces the tumor like enlargement.

Wens occur fairly commonly on the face, scalp, nucha and back but they may appear on any part of the body where sebaceous glands are present. Multiple cysts are not uncommon on the scrotum.

The *lesion* consists of a rounded mass usually up to the size of a walnut, firm but not hard and freely movable except at its point of attachment to the skin. When secondary pyogenic infection occurs with redness, swelling, pain and tenderness the cyst contents become an admixture of fluid, pus and cheesy material.

Cancerous degeneration in sebaceous cysts is not uncommon and is especially frequent in lesions of long duration. In almost every instance squamous cell carcinoma develops. It is perhaps the wisest procedure to have a pathologic examination made of the cyst after its removal.

Treatment—In all areas except the face *excision of the cyst* (p. 3935) is the simplest procedure. This can be done with infiltration anesthesia but care must be observed to remove the entire capsule. Where pyogenic infection is present incision and drainage only must be done. Radical cure is not attempted until the infection is controlled.

Excision can also be practiced in sebaceous cyst of the face but for cosmetic reasons other methods are more desirable. A simple and frequently successful procedure is to make a *small opening* into the top of the cyst under infiltration anesthesia, *express the cyst contents* and introduce a small *dermal curet* and scrape away the cyst lining. Care must be taken not to puncture the cyst wall. If more than half the lining wall is ablated cure will frequently result. In some instances of sebaceous cyst of the face cure has been obtained by the injection of 0.5 to 1.0 cc. of 70 per cent *alcohol* into the cyst after expressing the contents.

SYNOVIAL CYST

Synovial cysts are rare, rounded, translucent swellings occurring over the dorsal aspect of the *small joints* of the *fingers*. They are usually single and are believed to be extrusions of the synovial lining of the articulation. They contain yellowish fluid and are generally symptomless.

Satisfactory therapeutic results have been achieved with *roentgen therapy*. Recurrence is frequent after attempts at surgical excision.

MULTIPLE BENIGN CYSTIC EPITHELIOMA

Multiple benign cystic epithelioma is a rare disease in which *multiple small tumors* appear on the *face*. The condition is *familial*, affects *females* almost exclusively and generally makes itself noticeable shortly after puberty.

The *lesions* are distributed mainly in the *mid-portion of the face* about the root of the nose, the eyelids and the cheeks but in some instances may also appear on the trunk and arms. The neoplasm is more or less uniform and symmetrical about the size of a lentil, firm, slightly elevated and yellowish, pearly or pink in color. Over the surface may course *tatic*

vessels The lesions increase gradually to a maximum size after which they remain static

The history multiplicity and character of the lesions usually aid in differentiating this condition from basal cell epithelioma though in rare instances these lesions have undergone malignant transformation

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Blue Nevi (p 3 05)

Senile Sebaceous Adenomas (p 3 05)

Fibromas (p 3206)

Multiple Neurofibromatosis (*von Recklinghausen's Disease*) (p 3206)

Multiple Benign Cystic Epitheliomas (p 3 08)

Cerebroid Mole (p 3205)

Sebaceous Cyst (p 3 08)

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Wens occur fairly commonly on the face, scalp, nucha and back but they may appear on any part of the body where sebaceous glands are present. Multiple cysts are not uncommon on the scrotum.

The *lesion* consists of a rounded mass usually up to the size of a walnut, firm but not hard and freely movable except at its point of attachment to the skin. When secondary pyogenic infection occurs with redness, swelling, pain and tenderness the cyst contents become an admixture of fluid, pus and cheesy material.

Cancerous degeneration in sebaceous cysts is not uncommon and is especially frequent in lesions of long duration. In almost every instance squamous cell carcinoma develops. It is perhaps the wisest procedure to have a pathologic examination made of the cyst after its removal.

Treatment—In all areas except the face *excision of the cyst* (p. 5935) is the simplest procedure. This can be done with infiltration anesthesia but care must be observed to remove the entire capsule. Where pyogenic infection is present incision and drainage only must be done. Radical cure is not attempted until the infection is controlled.

Excision can also be practiced in sebaceous cyst of the face but for cosmetic reasons other methods are more desirable. A simple and frequently successful procedure is to make a *small opening* into the top of the cyst under infiltration anesthesia, *express the cyst contents* and introduce a small *dermal curet* and scrape away the cyst lining. Care must be taken not to puncture the cyst wall. If more than half the lining wall is ablated cure will frequently result. In some instances of sebaceous cyst of the face cure has been obtained by the injection of 0.5 to 1.0 cc. of 70 per cent alcohol into the cyst after expressing the contents.

SYNOVIAL CYST

Synovial cysts are rare, rounded, translucent swellings occurring over the dorsal aspect of the *small joints* of the fingers. They are usually single and are believed to be extrusions of the synovial lining of the articulation. They contain yellowish fluid and are generally symptomless.

Satisfactory therapeutic results have been achieved with *roentgen therapy*. Recurrence is frequent after attempts at surgical excision.

MULTIPLE BENIGN CYSTIC EPITHELIOMA

Multiple benign cystic epithelioma is a rare disease in which *multiple small tumors* appear on the face. The condition is *familial*, affects *females* almost exclusively and generally makes itself noticeable shortly after puberty.

The *lesions* are distributed mainly in the *mid portion* of the face about the root of the nose, the eyelids and the cheeks but in some instances may also appear on the trunk and arms. The neoplasm is more or less uniform and symmetrical about the size of a lentil, firm, slightly elevated and yellowish pearly or pink in color. Over the surface may course *telangiectatic*

| | |
|---|--|
| Granuloma pyogenicum | Proud flesh surrounding chronic staphylococcal infection. Usually of nails |
| Helminthiasis | Invasion of subcutaneous tissues by microfilaria as in filariasis, loiasis, onchocerciasis and dracunculosis. Identify parasite in blood stream, lymphatics or local lesion (p. 537) |
| Idiopathic multiple hemorrhagic sarcoma | Yasuni disease. Purplish nodules on foot and ankle. In elderly males (p. 3226) |
| Leprosy | Anesthetic nodules with ulceration. Often seen on face. Look for thickening of peripheral nerves, particularly ulnar branch at elbow. Get biopsy (p. 273) |
| Leukoplakia | White plaques with fissures. Often seen on buccal mucosa, lips, tongue, penis or vulva. A precancerous. Get biopsy (p. 3213) |
| Lymphosarcoma | Subcutaneous malignancy. Get biopsy (p. 3227) |
| Melanocarcinoma | Highly malignant cutaneous neoplasm with pigmentation. Early metastasis by satellite extension or through lymph channels (p. 3225) |
| Milium | Groups of rounded white nodules. Usually seen on face of young people. May involve genitals. Prick nodules and expel contents (p. 3403) |
| Moles | Pigmented or nonpigmented hairy giant, linear, cerebriform and sebaceous nevi (p. 3204) |
| Molluscum contagiosum | Umbilicated pearly tumors caused by virus infection. Most frequently seen in childhood (p. 3287) |
| Multiple benign cystic epitheliomas | Rare familial disease of the female. Lentil sized tumors of face. Get biopsy (p. 3208) |
| Necrobiosis lipoidica diabetorum | Yellowish nodular infiltrations of anterior aspects of thighs of diabetics. Probably related to insulin injections |
| Oroya fever | Crops of wartlike nodules due to infection with Bartonella bacilliformis (p. 384) |
| Rheumatic fever | Subcutaneous nodules. Often observed around elbows. Note accompanying arthralgia and carditis (p. 186) |
| Rheumatoid arthritis | Subcutaneous nodules very similar to those of rheumatic fever. Accompanied by ankylosing arthropathies but no carditis (p. 2910) |
| Sebaceous cysts | Usually single tumors containing caseous material. Treat by excision (p. 3206) |
| Steatocystoma multiplex | Skin colored tumors of anterior portion of trunk. Express sebaceous material by puncture (p. 3207) |
| Syringocystoma | Multiple small cystic tumors over anterior surface of trunk. In young females (p. 3209) |
| Synovial cysts | Translucent swellings over dorsal aspect of small joints of fingers. Often seen in osteoarthritis (p. 2855) |

 DIFFERENTIAL DIAGNOSIS OF

Dermatoses Characterized by Nodules, Cysts and Tumors

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|---------------------|---|
| Angiomas | Cavernous hemangiomas (strawberry mark) hemolymphangiomas and enile angiomas (p 3200) |
| Benign neoplasms | Fibromas glomus tumors lipomas neuromas, neurofibromas and adenomas Note pain with glomus tumors Multiple lipomas in adiposis dolorosa Multiple neurofibromas with pig- mentation in von Recklinghaus disease (p 3206) |
| Bowen's disease | Cutaneous malignancy of elderly patients Ulcerating papules of trunk and extremities Get biopsy (p 3225) |
| Calcinosis | Calcium depositions of skin. Demonstrate by x ray Note high blood calcium and associated scleroderma |
| Cutaneous horns | Yellowish black or brown papules in elderly patients A precancerosis (p 3217) |
| Dermatomycoses | Particularly deep fungous infections of ac- tinomycosis blastomycosis coccidioidomy- cosis and chromoblastomycosis Note indolent ulcerating granulomas Identify pathogen by smear or culture (p 40) |
| Epithelioma | Basal or squamous-cell variety (rodent ulcer or prickle cell malignancy) Former on face above upper lip Note waxy nodule ulceration, rolled border telangiectases and central crust Get biopsy (p 3935) Prickle cell epithelioma may involve ears lower lip tongue dorsum of hand or genitals Look for raised tumor with local infiltration ulceration and adherent crust Get biopsy (p 3220) |
| Erythema multiforme | Outbreak in spring and autumn between the ages of 10 and 30 Note constitutional symp- toms and distribution on dorsum of hands and feet and extensor surfaces of forearms and legs Look for iris-colored nodules in addition to vesicles and bullae Mucosal lesions often coexist (p 3374) |
| Erythema nodosum | Tender red or blue nodules on extensor sur- faces of legs Associated with constitutional symptoms (p 3377) |
| Ganglion | Fluctuant swelling involving tendon sheath. Usually of wrist (p 3296) |
| Gout | Tophi containing sodium urate crystals Us- ually located on hands feet and helix of ear Get blood uric acid Examine crystals micro- scopically (p 2867) |
| Granuloma fungoides | Fatal dermatosis characterized by itching scaling nodule and tumor formation with ul- ceration Get biopsy (p 3380) |

3216) In the majority of these instances a precancerous disposition exists and the mechanical trauma is then the precipitating factor

In the condition of *leukoplakia* mechanical irritation seems to stimulate the cells to assume malignant characteristics

Leukoplakia—The leukoplakial lesion is of variable size and consists of a well defined grayish or silvery white slightly elevated area of the mucosa of the cheeks lips tongue penis or vulva The surface of the leukoplakial plaque is smooth or somewhat roughened In some instances the patches become thickened and infiltrated *Fissures erosions or ulcers* may appear as the result of the trauma The presence of any of these secondary changes or of *advancing growth or infiltration* is of ominous portent frequently signifying *malignant degeneration* and the production of a *squamous cell carcinoma* Though the local mucosal changes are fairly common the assumption of the characteristics of malignancy is relatively rare

Exciting Causes—Leukoplakias rarely are encountered under normal circumstances Malocclusion absent teeth badly fitted dentures smoking the irritation of a broken pipe chronic gingivitis carious and jagged teeth and the friction of wads of chewing tobacco constitute important contributory factors in the mouth Though syphilis was once thought to be an inevitable accompanying circumstance in buccal leukoplakia the association is now known to be less frequent than was believed

Diagnosis—Leukoplakia of the buccal cavity may resemble *lichen planus* and *lupus erythematosus* The latter disturbance should offer little difficulty since there is usually an accompanying lesion of the glabrous skin and the individual lesion is much redder and may be atrophic it more often affects the lip than the mucosa of the cheek The eruption of lichen planus has a tendency to be reticulated forming a mosaic pattern and is not necessarily localized to an area of chronic irritation It occurs further back in the mouth on the buccal mucosa than is usual with leukoplakia In addition cutaneous lesions of lichen planus are usually present See Fig 921

Treatment—The presence of leukoplakial lesions requires a search for the provocative irritant Smoking and the use of chewing tobacco should be stopped completely The teeth and the gums should be treated by a competent dentist The patient is instructed to report for frequent inspection and is instructed to avoid spicy or irritant foods and liquids in the diet

The physician and dentist are to avoid the use of *caustics* for the treatment of the individual lesion Silver nitrate particularly must not be used for it tends to stimulate epithelial proliferation and may hasten carcinomatous change If there is a suspicion of malignant degeneration a section of tissue from the most suspicious area should be removed and a histologic study made If there are evidences of a squamous-cell carcinoma the patient is referred to the *specialist for wider excision and roentgen therapy*

Radiant Energy—There is an obvious and unfortunate relationship between malignant degeneration and exposure to solar and radiant energy Thus the extraordinary congenital familial condition of *xeroderma pigmentosum* (p 3158) may reveal malignant degeneration of its lesions upon exposure to sunlight or artificial sources of actinic rays Carcinomatous

Syphilis

Nodulo-ulcerative syphilids forming geometric patterns with positive serology (p 336)
Gummas with punched out ulcers Therapeutic response to iodide and penicillin even in the presence of negative serology (p 3286)

Tuberculosis

Tuberculosis verrucosa cutis with circumscribed wart like patches on dorsum of hands (p 3259) Scrofuloderma of skin overlying caseous lymph node (p 3262) Lupus vulgaris of cheek or nose with apple jelly nodules ulceration and destruction of tissue (p 3267) Erythema induratum (Bazin) with chronic necrotizing nodules usually of calves in women (p 3271) Sarcoidosis with brownish nodules and plaques especially of the face Note accompanying involvement of ophthalmic structures (p 3271) Get biopsy in all instances

Verrucae

Warts of pin head to pea size May involve hands feet or perineal regions Latter usually venereal in uncleanly individuals Make local examination by dark field spread and culture Get Wassermann and Frei tests (p 472) Plantar warts often exceedingly painful Note therapeutic response to x ray or radium

Xanthoma

Yellow papules of eyelids arms elbows axilla neck or cornea (pinguecula) Usually associated with diabetes mellitus or reticulo-endotheliosis (p 1137) Check blood sugar hemogram and marrow smears (p 1035)

Multiple growths cannot be attacked surgically for practical reasons but any one of the growths which shows evidence of growth or ulceration should be eradicated and studied Should sections show evidence of malignancy prophylactic roentgen therapy should be inaugurated for the destruction of nests of cells that may have been missed in the surgical procedure

Patients who have any of the precancerous conditions which involve multiple growths should be warned against cutaneous irritation in any of the forms later to be described (p 3212) Exposure to the carcinogenic agencies is to be avoided by patient and physician These include mechanical irritation photosensitization the use of chemicals particularly tar and arsenic and the administration of estrogen

MALIGNANCY ARISING IN DERMATOSES CAUSED BY CHRONIC IRRITATION OR INFECTION

The production or perpetuation of a dermatosis by chronic irritation and infection may result in malignant degeneration The carcinogenic factor may be mechanical radiant infectious or chemical

Mechanical Trauma—Mechanical trauma that is persistent and long standing may cause malignant degeneration in any of the benign neoplasms (p 3199) It also is a contributory factor in degeneration that occurs in scars (especially those due to burns) that are subjected to friction and many of the keratotic lesions associated with gonadal decreas (p

| | LESION | SITE OF PREDILECTION | CLINICAL AND DIAGNOSTIC FEATURES |
|---------------------------|---|--|--|
| Pruritus hiemalis | None | Generalized | Winter itch (p 3171) |
| Seborrheal keratosis | Pigmented tumors | Scalp face and trunk | Greasy scales (p 3217) |
| Squamous cell epithelioma | Raised tumor with ulceration and crusting | Lower lip ear tongue hands or genitals | Malignancy Get biopsy (p 3223) |
| Vulvovaginitis | Dryness with atrophy and fissures | Genitals | Itching Precancerous Get biopsy (p 2-86) |

degeneration is also observed particularly in older men in *chronic actinic dermatitis* the so called *farmers or sailors skin* (p 3175)

Of greater frequency and importance is the cancerous degeneration of areas of skin afflicted with chronic radiodermatitis (p 3179) In the earlier days of radiation treatment many of the most prominent roentgenologists lost fingers and hands as the result of carcinomatous change resulting from excessive exposure to the ray

Infection—The persistence of chronic infection may occasionally be a carcinogenic factor Thus a *chronic ulcer* no matter where situated may develop degeneration at its margin and become carcinomatous Under these circumstances sections usually show *squamous cell carcinoma* This unfortunate sequence of events has been observed in traumatic ulcers of the skin and mucous membranes decubitus ulcers and those due to severe burns radiodermatitis and varicosities in the veins of the legs

Malignant degeneration also occurs in the lesions of *syphilis* (p 3278) Leukoplakia due to syphilis may develop malignancy Syphilitic interstitial glossitis and gumma of the tongue uncommon conditions in these days of modern syphilotherapy may also be the precursors of squamous-cell carcinoma This change is usually preceded by the development of an indurated nodular mass which undergoes ulceration The physician must not be lulled into dangerous apathy by the presence of a positive Wassermann test of the blood and be satisfied to proceed merely with antisiphilitic therapy Immediate *biopsy* should be performed and if the presence of squamous cell carcinoma is demonstrated proper treatment by *surgery and radiation therapy* instituted

Chemical Irritation—Carcinogenic activity may be produced in the skin by the local or systemic use of certain drugs or chemicals employed in industry and medicine

The carcinogenic properties of certain chemicals have been well shown in animal experiments and by the statistical surveys among workers exposed to these substances Of chief importance are *tar tar distillates pitch aniline dyes and dye intermediates soot petroleum machine oils and arsenical compounds* (Paris green arsenical plant sprays)

Occupational Keratoses—A variety of changes are encountered in the skin of workers with carcinogenic substances but most important are the

DIFFERENTIAL DIAGNOSIS OF

Common Dermatoses of the Aged

| | LESION | SITE OF PREDILECTION | CLINICAL AND DIAGNOSTIC FEATURES |
|---|------------------------------|--|---|
| Acroparesthesia | None | Hands and feet | Abnormal sensations Usually in elderly women (p 3230) |
| Alopecia | Baldness | Scalp | Loss of hair |
| Angioma | Isolated red tumors | Face and trunk | Vascular character (p 3200) |
| Atrophy | Wrinkling | Generalized | Loss of elasticity |
| Basal cell epithelioma | Waxy nodule (rodent ulcer) | Middle third of face usually above upper lip | Telangiectases Rolled edge Ulceration. Malignancy Get biopsy (p 3220) |
| Bowen's disease | Grouped eroded papules | Trunk and extremities | Ulceration Malignancy Get biopsy (p 3225) |
| Chilblains | Frostbite | Digits nose and ears | Vesiculation and gangrene Consider use of anticoagulants |
| Cutaneous horn | Yellow brown or black tumors | Head and trunk | Biopsy (p 3217) |
| Decubitus ulcer | Bed sore | Sacrum, elbows and heels | Delayed healing |
| Dermatitis hemorrhagica | Pigmentation and ulceration | Shins | Secondary to varicose veins |
| Gout | Tophi | Ears fingers and elbows | Sodium urate crystals |
| Idiopathic multiple hemorrhagic sarcoma | Purple nodules | Foot and ankle | Malignancy Get biopsy (p 3226) |
| Keratoderma climactericum | Fissures and hyperkeratoses | Palms and soles | Treat with estrogen (p 3240) |
| Keratoses | Pigmented spots with scaling | Face chest and backs of hands | Precancerous Get biopsy (p 3166) |
| Neurodermatitis | Flat, itching papules | Neck and extremities | In females Psychogenic |
| Leukoplakia | White plaques | Buccal mucosa lips tongue and genitals | Precancerous Get biopsy (p 3213) |
| Paget's disease of breast | Dermatitis | Nipple and areola | Malignant. Get biopsy (p 2581) |

question of beginning change into cancer. If there are a great many lesions only the suspicious ones need be treated while the others are observed at regular intervals for evidence of degeneration.

The simplest method of removal is by thorough *electrodesiccation* and *curettage* under infiltration anesthesia. Roentgen therapy is also effective.

Seborrheal Keratosis (Senile Wart)—The seborrheic keratosis (senile wart, seborrheic wart) is an elevated pigmented round or oval lesion which occurs most commonly on the *trunk*. The lesion occurs in both sexes more often in elderly persons but not uncommonly in the fourth or fifth decades. Its exact etiology is unknown but it is often seen in association with the seborrheal type of skin. Malignant degeneration apparently is rare and when it does occur is practically always into *basal cell epithelioma*.

Seborrheic keratosis may occur singly but usually there are multiple lesions at times over a hundred. Individually they are regular round or oval raised firm and tan to brownish black in color. The surface is *smooth* or *wart like* and may be covered with a *greasy scale*. The site of predilection is the anterior and posterior surface of the trunk (Fig. 922).

When but a few lesions are present it is a simple task to remove them. However in the presence of a great profusion of lesions it may be preferable to observe the patient and treat only those which show suspicious signs of degeneration. The simplest procedure is to *electrodesiccate* the lesion lightly and then scrape it away with a *curet*.

Cutaneous Horn (Cornu Cutaneum)—A cutaneous horn is a skin projection shaped somewhat like the horn of cattle and composed of thickened cornified epithelial cells. It is observed in elderly persons more often males. It may arise directly from the senile skin or from a keratotic area, a pathologic follicle or a sebaceous cyst. The lesion is most frequent on the face, scalp, male genitals and the extremities. It is usually single and consists of a yellowish brown or blackish brown firm horn shaped dry mass which may be as small as a pinhead or up to 2 inches in length.

The cutaneous horn is a precancerous lesion. More than 10 per cent show degeneration into *squamous cell epithelioma* at the base.

Treatment—Unless there is clinical evidence of malignant change the lesion is removed by *electrodesiccation* or *cauterization*. If there is any question of degeneration thorough *excision* should be practiced and the tissue studied by a *pathologist*. The suspicion of malignancy calls for prophylactic *roentgen treatment* (p. 3796).

Miscellaneous Malignant Degenerations—Malignant degeneration may also occur in other cutaneous afflictions. These include the rare disease of *erythroplasia* (p. 3381) and certain of the chronic cutaneous afflictions associated with atrophy such as *lupus erythematosus* (p. 3395), *lupus vulgaris* (p. 3260) and *acrodermatitis chronica atrophicans* (p. 3366).

MALIGNANT NEOPLASMS

Malignant neoplasms of the skin may be *primary* or *secondary*. The primary growths are the *carcinomas* which spring from the epithelial cells and the *sarcomas* which originate in the mesodermal structures. *Secondary deposits* may result from direct extension from a neighboring growth which has ulcerated into the skin or from lymphatic and hematogenous spread.

keratoses many of which develop into squamous cell carcinoma. The exposed areas of the face and hands are chiefly affected as a rule, but in chimney-sweep's cancer the scrotum is involved.

The most pressing indication in workers exposed to these hazards is for periodic physical examination and prophylactic measures. When the skin is already affected a *change of occupation* is required and *soothing applications* are applied. Keratoses should be removed by *excision* or *electrodesiccation*.

Arsenical Keratoses—Arsenical keratoses most frequent on the palms and soles may degenerate into squamous cell carcinomas. These lesions follow the prolonged administration of inorganic arsenic (Fowler's solution Asiatic Pills).

Prevention of this condition rests with the physician and pharmacist. Patients must not be allowed to take the inorganic arsenicals indefinitely. The drug must be given only for a reasonable time and preferably not used at all in chronic or recurrent diseases such as psoriasis. The keratoses may be removed by scalpel excision or electrodesiccation.

Estrogen—In experimental animals *estrogen has proven carcinogenic properties*. Whether this observation carries over into human malignancy is difficult to decide. Since estrogen is given mostly for symptomatic relief the practitioner is wise to avoid its use as much as possible until more data are available. The patient with a *precancerosis* or a family history of *malignancy* should be *dissuaded* from estrogenic therapy if possible.

MALIGNANT CHANGE IN LESIONS ASSOCIATED WITH GENITAL DEGENERATION

Malignant degeneration may occur in *senile keratoses*, *seborrheic keratoses* (senile warts, seborrheic warts), the *cutaneous horn* and *atrophy of the vulva* (kraurosis vulvae).

Atrophy of the vulva is more fully discussed elsewhere (p. 2595). It is sufficient to state here that untreated and unrelieved it is followed by squamous cell carcinoma in a high percentage of instances.

Senile Keratosis—The senile keratosis is a localized hyperkeratosis occurring principally on the *exposed parts* of the skin of elderly persons. It is seen chiefly on the face, the back of the hands and the forearms. It may occur on the lip, especially the lower and in this situation is especially likely to terminate in *squamous cell carcinoma*. The lesion is seen almost exclusively in the *white race*, the dark-skinned peoples being nearly entirely exempt. It occurs in elderly persons, especially those who have been exposed to strong sunlight over a long period of time.

As a rule multiple lesions are present. They consist of *pigmented spots* irregular in outline and varying in size up to a lima bean. The surface is roughened and covered with an adherent scale which may be *dirty gray*, *dull brown* or *yellow*. The lesions gradually become more elevated and thickened. At times the surface becomes verrucous. Ordinarily there is no redness present about the lesion, but if there is concomitant elevation its appearance suggests malignant change.

The condition most commonly eventuates in *squamous cell carcinoma* but may form neoplasms of the basal-cell variety.

Senile keratoses of the lip should be eradicated as soon as the diagnosis is made. They should be removed from the glabrous skin if there is any

question of beginning change into cancer. If there are a great many lesions only the suspicious ones need be treated while the others are observed at regular intervals for evidence of degeneration.

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 DIFFERENTIAL DIAGNOSIS OF

Fissures, Cracks, Clefts, Rhagades and Ulcers of the Skin

Dermatoses associated with interruption of skin continuity are invariably complicated by scabs, crusts and scarring. The secondary features often obscure the more fundamental lesion.

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|------------------------|--|
| Actinomycosis | Indolent granulation with ulceration. Isolate ray fungus (p 489) |
| Anthrax | Malignant pustule. Often with surrounding vesicles. Identify gram positive spore bearing bacillus (p 292) |
| Anobioflavinosi | Rhagades particularly at angles of mouth. Note therapeutic response to vitamin B complex (p 623) |
| Atopic dermatitis | Infantile eczema. Seek offending allergen, often a digestant like milk or eggs |
| Burns | Due to heat or chemicals. Check history. May be painless in anesthetic areas |
| Chancroid | Venereal infection, producing soft chancre. Get dark field to eliminate syphilis. Identify <i>H. ducreyi</i> (p 288) |
| Chapping | Superficial fissures due to moistening of lip in cold weather |
| Chemical dermatitis | Resulting from exposure of skin to irritants |
| Decubitus ulcer | Over pressure points such as sacrum, elbows, heels and occiput. In aged and debilitated patients (p 3167) |
| Dermatitis hemostatica | Varicose vein ulcer complex (p 3371) |
| Dermatomycoses | Deep fungous infections. Identify organisms producing blastomycosis, coccidioidomycosis, chromomycosis, Madura foot, sporotrichosis, aspergillosis or torulosis (p 3298) |
| Epithelioma | Basal cell lesions of face above upper lip. Prickle cell variety of ears, lower lip, hands or genitals. Note indolent ulcer with rolled edge and adherent crust. Get biopsy (p 3220) |
| Erosive balanitis | Fusospirochetal infection of penis. Refer to specialist for darkfield microscopy. Get serology (p 2456) |
| Feigned eruptions | Excoriations of forearms due to scratching. A neurosis (p 3232) |
| Foot and mouth disease | Virus infection productive of ulcerating vesicles of oral mucous membrane and extremities (p 437) |
| Frambesia (Yaws) | Spirochetal infection due to <i>treponema pertenue</i> . Note extragenital crusted ulcer at point of inoculation. Check dark field and serologic tests. Note prompt response to penicillin (p 351) |
| Granuloma fungoides | Fatal dermatosis beginning with simple dermatitis and progressing through stages of nodule formation and fungating tumors. Get biopsy (p 3386) |

| | |
|---|---|
| Granuloma inguinale | Venereal infection. Identify Donovan bodies in smears or by biopsy (p 475) Exclude syphilis by darkfield microscopy |
| Helminthiasis | Productive of ulcerations in tropical areas Including blinding filaria eye worm and dracunculus Identify microfilaria or nematodes (p 537) |
| Hemorrhagic diseases | Ulcerating lesions observed in sickle cell anemia congenital hemolytic anemia Hodgkin's disease and leukemia Study hemogram. Get biopsy of local lesion (p 1108) |
| Idiopathic multiple hemorrhagic sarcoma | Kaposi's disease Purplish nodular lesions of legs with ulceration Get biopsy (p 3226) |
| Keratoderma climactericum | Keratosis and fissuring of palms and soles at time of menopause (p 3240) |
| Leprosy | Anesthetic ulcers of face nose and extremities Get biopsy (p 273) |
| Leukoplakia | Precancerous with white plaques and fissures of buccal mucous membrane tongue or genitals Get biopsy (p 3213) |
| Lymphopathia venereum | Venereal infection with transitory initial lesion that ulcerates Note regional lymphadenopathy and hypersensitivity in Frei test (p 471) Exclude syphilis by dark field microscopy |
| Paget's disease of the breast | Ulcerating eruption of nipple and areola Associated with malignancy of the breast Get biopsy and refer to surgeon for radical mastectomy (p 2581) |
| Pemphigus | Fatal bullous dermatitis with denudation and ulceration Get biopsy (p 3406) |
| Perforating ulcers | Usually of toe In peripheral arteriosclerosis thromboangiitis-obliterans and peripheral neuritis due to diabetes tabes dorsalis or syringomyelia Note local vascular and neurologic status Obtain Wassermann test |
| Protozoal diseases | Particularly American and Oriental leishmaniasis producing chronic tropical ulcers Identify leishmania in smears (p 506) |
| Radiodermatitis | History of x ray or radium therapy Note atrophy telangiectasia and ulceration (p 3160) |
| Rat bite fever | Chancre at point of inoculum. Attempt to identify treponemas Get history of rat bite (p 363) |
| Senile vulvovaginitis | With local atrophy fissuring and precancerous changes Accompanied by intense pruritus Get biopsy (p 2597) |
| Syphilis | Hard chancre with positive dark field (p 3278) Nodulo-ulcerative secondary syphilids with positive dark field and serology (p 3281) Gumma with ulceration and therapeutic response to iodide (p 3286) |
| Tularemia | Necrotic papule at site of inoculum History of bite by rodent (p 323) |
| Tuberculids | Chancre scrofula papulonecrotic tuberculid lupus vulgaris erythema induratum or sarcoidosis Get biopsy (p 3255) |

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|------------------------|--|
| Actinomycosis | Indolent granulation with ulceration. Isolate ray fungus (p 489) |
| Anthrax | Malignant pustule. Often with surrounding vesicles. Identify gram positive spore bearing bacillus (p 292) |
| Arboflavinosis | Rhagades particularly at angles of mouth. Note therapeutic response to vitamin B complex (p 623) |
| Atopic dermatitis | Infantile eczema. Seek offending allergen, often a digestant like milk or eggs |
| Burns | Due to heat or chemicals. Check history. May be painless in anesthetic areas |
| Chancroid | Venereal infection producing soft chancre. Get dark field to eliminate syphilis. Identify <i>H. ducreyi</i> (p 288) |
| Chapping | Superficial fissures due to moistening of lip in cold weather |
| Chemical dermatitis | Resulting from exposure of skin to irritants |
| Decubitus ulcer | Over pressure points such as sacrum, elbows, heels and occiput. In aged and debilitated patients (p 3167) |
| Dermatitis hemostatica | Varicose vein ulcer complex (p 3371) |
| Dermatomycoses | Deep fungous infections. Identify organisms producing blastomycosis, coccidioidomycosis, chromophytosis, Madura foot, sporotrichosis, aspergillosis or torulosis (p 3298) |
| Epithelioma | Basal cell lesions of face above upper lip. Prickle cell variety of ears, lower lip, hands or genitals. Note indolent ulcer with rolled edge and adherent crust. Get biopsy (p 3220) |
| Erosive balanitis | Fusospirochetal infection of penis. Refer to specialist for darkfield microscopy. Get serology (p 2456) |
| Feigned eruptions | Excoriations of forearms due to scratching. A neurosis (p 3232) |
| Foot and mouth disease | Virus infection productive of ulcerating vesicles of oral mucous membrane and extremities (p 437) |
| Frambesia (Yaws) | Spirochetal infection due to <i>treponema pertenue</i> . Note extragenital crusted ulcer at point of inoculation. Check dark field and serologic tests. Note prompt response to penicillin (p 351) |
| Granuloma fungoides | Fatal dermatosis beginning with simple dermatitis and progressing through stages of nodule formation and fungating tumors. Get biopsy (p 3386) |

Rodent Ulcer—The rodent ulcer may arise from normal skin or at the site of a keratosis comedone or other benign lesion. The growth occurs



Fig 9.5—Bowen's disease

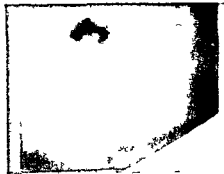


Fig 9.6—Melanocarcinoma (with distant metastases)



Fig 9.7—Melanocarcinoma (with satellite extension)

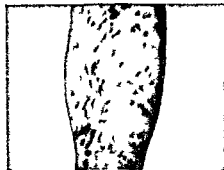


Fig 9.8—Idiopathic multiple hemorrhagic sarcoma (Kaposi)



Fig 9.9—Idiopathic multiple hemorrhagic sarcoma (Kaposi)



Fig 9.10—Lymphoblastoma

predominantly on the face but occasionally attacks the trunk or extremities. The area of predilection is the region above the upper lip in the middle portion of the face. A single growth is the rule but multiple tumors

from a remote primary growth. Secondary growths have the pathologic characteristics of the tumor from which they are an offshoot.

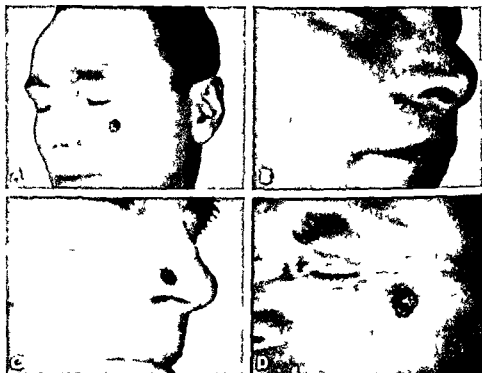


Fig 923—A B C D Basal cell epithelioma (rodent ulcer)

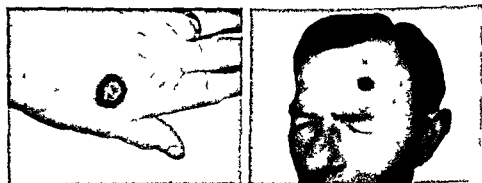


Fig 924—Squamous cell epitheliomas (prickle cell)

BASAL CELL EPITHELIOMA (RODENT ULCER)

The basal cell epithelioma is one of the commonest malignant growths encountered and although it does not metastasize it may cause profound local destruction of tissue. It is more frequent in men than in women and usually appears after the fiftieth year though many examples are seen in younger persons. It often develops in the lesions of *xeroderma pigmentosum* in childhood.

cinoma In these instances the therapy recommended for squamous cell epithelioma should be employed (p 3223)

SQUAMOUS CELL EPITHELIOMA (PRICKLE CELL EPITHELIOMA)

The squamous cell epithelioma is a *malignant metastasizing* form of carcinoma arising primarily in the skin and adjacent mucous membranes It affects *males* far more frequently than females and is most common in adult life between the fortieth and sixtieth year It may arise from apparently normal skin or mucous membrane but more often it is a malignant degenerative change in some pre-existent lesion Thus it may develop in the various keratoses leukoplakia ulcers scars cutaneous horns and other forms of precancerosis (p 3209)

The prickle cell epithelioma is a raised firm *wart-like* growth or a flat localized area of *infiltration* at the onset *Ulceration* soon ensues with the formation of an adherent crust Palpation reveals the *deep induration* of the lesion and its extension into the subjacent tissues At times there is proliferation of the lesion and its surface becomes *fungoid* or *verrucous* with a malodorous discharge The lesion occurs most frequently on the ears lower lip tongue buccal mucosa dorsum of the hands the glans penis and the vulva It is practically invariably a solitary tumor and tends to extend fairly rapidly and cause destruction of the tissues In a few months it can attain a size which a basal cell epithelioma needs years to reach Untreated there are metastases to the regional lymph nodes the internal viscera and bones Death is usually the result of cachexia due to generalized metastases

The *diagnosis* of squamous cell epithelioma can be made clinically but must always be confirmed by histologic study

The *treatment* of squamous cell carcinoma requires consultation with the *surgeon* and *radiotherapist* The various modalities employed include (1) *scalpel surgery* (2) *cutting current* (3) *electrocoagulation* (4) *roentgen therapy* These are used singly or in combination depending upon the size location and depth of the lesion the age of the patient and the presence or absence of metastatic lesions

PAGET'S DISEASE OF THE BREAST

Paget's disease is an unusual condition of the *female breast* in which there appear simultaneous *carcinomas of the skin and the mammary ducts* This condition was once included as a precancerosis but it is now generally agreed to be a cancer from its inception Rare instances have been observed in the male breast and also in locations outside of the breast (extra mammary Paget's disease) It is more frequent in women past forty but has been seen in younger persons

Clinically Paget's disease presents an *eczema-like eruption of the nipple and areola* with extension onto the skin of the breast This *eczematoid* eruption is usually well defined and may be dry red and scaly or edematous oozing eroded and crusted The nipple may be normal or it may appear thickened and crusted or retracted Generally subjective symptoms of burning itching tingling or dull pain are present Probably in every case of Paget's disease of the breast there is an associated adenocarcinoma of the mammary duct

may be present. Primary growths of the mucosa probably never occur but there may be direct extension from the skin near the lips or nares.

A number of different clinical varieties are observed. The most frequent is the *nodular type* in which a group of small rounded coalescent nodules appears. These are firm and waxy or pearly. As the growth enlarges central destruction of the nodules with *ulceration* and *crust formation* occurs. At this stage the nodular border of the lesions assumes a very characteristic appearance being rolled raised and pearly. *Telangiectatic vessels* may be present over the tumor. The central crust becomes detached or is picked away and exposes a moist bleeding ulcerous depression. The latter may gradually increase in depth as a new and deeper crust continually reforms. The lesion grows slowly and may be no larger than a dime after several years. In some instances growth is more rapid and ulceration proceeds deeply and widely. These lesions may attack and destroy the bone and cartilage the eyelid the eye and any other structure adjacent to them. Secondary pyogenic infection may supervene with local or general sepsis and severe hemorrhage may ensue from erosion of a large blood vessel. These are the cases which may have a fatal outcome.

Other clinical appearances may be present in basal cell epithelioma. The lesion may become *papillary* or *verrucous* resembling a large wart or granuloma due to syphilis tuberculosis or a drug eruption (bromides iodides). Pigmented flat and atrophic types are recognized but occur rarely.

Multiple Epitheliomatosis—Superficial or multiple epitheliomatosis is an unusual variant in which there are multiple lesions on the trunk especially the back though they may appear in other situations. They consist of irregular flat superficial well demarcated *plaques* of a deep red to bronze color. The patch is usually dry and smooth but may be scaly or moist and crusted. The border of the lesion is often raised pearly and threadlike.

The lesions grow slowly over a period of years but at times may lead to invasive and destructive ulcers. In some instances of superficial epitheliomatosis there is a history and other evidence of prolonged ingestion of inorganic arsenic. It is probable that arsenic may be an etiologic factor in these.

Diagnosis—The diagnosis of basal cell epithelioma can generally be made by inspection. The location and *chronicity* of the lesion the characteristic *rolled pearly border* the *age* of the patient and the *slow evolution* are important diagnostic criteria. However it is recommended that a *biopsy* be made in every instance to confirm the diagnosis and especially to eliminate the possibility that the lesion is a squamous cell carcinoma or a mixed type of growth.

Treatment—The treatment of basal-cell epithelioma depends upon the age of the patient and the location size and depth of the growth. The procedures that have been used with success are (1) *scalpel excision* (2) *curettage* (3) application of *acid nitrate of mercury* (4) *electrodesiccation* or *electrocoagulation* (5) *carbon dioxide snow* (6) *roentgen therapy*. These modalities are best handled by the expert who can decide which is the most suitable method for the individual case.

When the lesion is refractory to therapy consideration must be given to the possibility that the growth is a mixed basal and squamous cell car-

Cysts and Neoplasms

Congenital cyst (unilateral with occasional discharge of witch's milk) (p 2778)
 Chronic cystic mastitis (multiple cysts with evidence of inflammation) (p 2580) Galactoceles (during lactation) Blue dome cysts (solitary lesions appearing just before or just after menopause) Benign fibromas and lipomas (p 2579) Intracanalicular fibromas (p 2581) Carcinoma and sarcoma of breast (p 2581) Biopsy

Sebaceous adenoma of nipple (p 3149) Epithelioma of nipple (p 3208) Paget's disease of the nipple (p 2581) Biopsy

Differential diagnosis must be made from *seborrheic eczema* (p 3432) or *dermatitis venenata* (p 3330) These conditions usually respond to proper local therapy in a reasonable period If there is not the anticipated improvement *biopsy* should be performed

Treatment combines *radical mastectomy* and *roentgen therapy*

BOWEN'S DISEASE

Bowen's disease is a rare condition at one time deemed a precancerosis but now generally conceded to be from its incipency a form of intra-epidermal carcinoma It attacks the sexes equally and usually appears in patients past the age of forty years (Fig 925)

The lesions of Bowen's disease have been noted principally on the trunk and extremities but may also occur on other areas and even on the mucous membranes There may be one or more lesions which appear as crusted eroded red *papules* The papules coalesce and enlarge to form infiltrated *plaques* covered with crusts and scales or at times with a verrucous surface Bowen's disease eventuates in squamous cell epithelioma in the great majority of instances less often in basal cell epithelioma

Differential diagnosis is not easy on a clinical basis *lupus vulgaris* (p 3262) *tertiary syphiloderms* (p 3286) and *basal-cell epithelioma* (p 3220) having a great similarity Usually *histologic study* is required for confirmation

The most successful forms of *treatment* are *excision* *electrodesiccation* or *electrocoagulation* Roentgen therapy is apparently not beneficial

MELANOCARCINOMA (MALIGNANT MELANOMA NEVOCARCINOMA)

Melanocarcinoma is an extremely malignant growth arising in a pigmented nevus It may develop spontaneously in a benign lesion but more often follows repeated irritation inflammation or injudicious therapy It may occur at any age but is most frequent in the third and fourth decades The most important precursor is the flat or raised smooth non hairy slate blue or bluish black nevus (p 3204) The majority of cases occur on the lower extremities especially about the toes but they also arise on the face the genitals and in the nail fold or nail bed (Hutchinson's melanotic whitlow)

The *lesion* consists of a single nodule or multiple nodules dark brown brownish black slate blue bluish black or black in color These may be

 DIFFERENTIAL DIAGNOSIS OF

Clinical Disturbances of Breasts and Nipples

The female breast may suffer physiologic disturbances particularly referable to lactation in pregnancy. The more common organic difficulties include acute infections usually the result of suckling and neoplastic involvements. The most serious of the latter is the malignant growth whose presence must be suspected in the presence of any palpable induration or in association with serosanguineous discharge from the nipple.

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|------------------------------------|---|
| Physiologic | Premenstrual and menstrual congestion and engorgement (p. 2489). Colostrum secretion and lactation during pregnancy and the puerperium (p. 2623). Atrophy after menopause. Hypertrophy during pregnancy and lactation. |
| Pharmacodynamic | Hypertrophy with estrogen (p. 2515). Lactation from lactogenic anterior pituitary hormone (p. 1154). Agalactea following pregnancy after estrogen or androgen (p. 2401). Malignant transformation from prolonged use of estrogen (p. 2515). |
| Secretory Disturbances | Witch's milk from congenital cysts in the non-pregnant (p. 2778). Lactation of the newborn. Agalactea, polygalactea and caked breasts in pregnancy (p. 2623). |
| Gynecomastia | Hypertrophy of male breast with lactation in hypopituitarism and dyspituitarism (Frohlich's syndrome) (p. 1166). |
| Tegumentary Disturbances | Pigmentation (pregnancy, lactation, adrenal cortical deficiency, hyperpituitarism and hyperthyroidism) (p. 1197). Lymphedema (peau d'orange) with advanced malignancy (p. 2581). Retraction of nipples with malignancy. Get biopsy. |
| Neurogenic Disturbances | Mastodynia or mastalgia from trauma (p. 2546). Engorgement of pendulous breasts. Irritation of ill-fitting brassieres. Premenstrual tension. Pregnancy. Lactation. Caking of breasts. Mastitis (p. 2612). Also in neuroses and hysteria. |
| Congenital Anomalies | Asymmetry. Accessory breasts or nipples in axilla or along mammary ridge between mid-clavicular region and inguinal fossa (p. 2533). Inversion of nipple (p. 2533). |
| Mechanical Disturbances and Trauma | Pendulous breasts. Acute ecchymosis or hematoma with traumatic fat necrosis (p. 2546). Chronic irritation from pressure of ill-fitting brassiere. Cracks and fissures of nipples (p. 2623). |
| Inflammations and Infections | Acute mastitis (p. 2612). Chronic mastitis (p. 2613). Chancre of nipple (p. 2613). Tuberculous mastitis (p. 2613). Check darkfield for spirochetes (p. 45) and biopsy for tuberculois. |

and they vary in size up to several inches. Ulceration and other secondary changes rarely occur (Fig. 928, 929).

Lesions may be confined to the lower extremities for many years or new ones may gradually appear elsewhere on the skin surface. In this manner a generalized distribution of the disease is produced. Sometimes *visceral involvement* develops early in the course of the disease or other times after many years. Any organ in the body may be affected but involvement is seen chiefly in the gastro-intestinal tract, liver, lungs and lymph nodes. It is not exactly clear whether visceral involvement is the result of metastasis or is due to the multicentric origin of the neoplasm in various tissues.

The exact etiology of this disease is unknown but some give credence to the belief that it is due to a low grade infectious agent. Others consider it a form of neoplastic affliction of the reticulo-endothelial system.

The *diagnosis* is simple if the condition is suspected. *Histopathology* is typical and should always be used to confirm the clinical diagnosis.

The *prognosis* must be guarded. In some patients there is a fatal outcome within a year while others have lived more than twenty-five years after the diagnosis was made.

Treatment consists of the administration of arsenic (Fowler's solution) orally and roentgen irradiation of the skin lesions.

METASTATIC CARCINOMA

Metastatic skin lesions occur in breast malignancy. They are often seeded along the tract of needle puncture resulting from chest aspirations for intrathoracic cancer. Rarely primary neoplasms of the internal viscera as hypernephroma may spread hematogenously and reach the skin. *Histologic study* is essential for final diagnosis.

LYMPHOSARCOMA

Lymphosarcoma is primarily a disease of the lymph nodes and may appear as an involvement of one group of regional lymph nodes or there may be simultaneous affection of various groups of nodes. In rare cases the skin reveals multiple firm painless bluish or brownish red flat or rounded tumors. The diagnosis is readily made by *histologic study* (Fig. 381).

Lymphosarcoma is very *radiosensitive* and responds promptly and well to roentgen rays. Recurrences are customary however and a fatal outcome is inevitable. Arsenic (Fowler's solution) is considered a beneficial adjuvant.

NEUROGENIC, AUTONOMIC AND PSYCHOGENIC DISTURBANCES OF THE SKIN

The peripheral nerves of the skin include the *sensory fibers* conveying the sensation of pain, touch, heat and cold and the *neurogenic mechanisms* of the involuntary nervous system which are distributed to the sweat glands, the blood vessels and the arrectores pilorum.

Organic and Functional Nervous Lesions.—As the result of alterations in the innervation of voluntary and involuntary systems, widespread disturbances occur in the tegumentary system. These are *organic* when atrophy of the skin follows degeneration of a sensory nerve. *Functional disturb-*

arranged as satellites about the parent growth or they may coalesce into a solid deeply colored *plaque*. Occasionally there occurs a melanocarcinoma without visible pigmentation (amelanotic melanocarcinoma) although pigment may be present in the histologic section. This form is similar in other respects to the pigmented variety. As a rule *metastasis to the regional lymph nodes* occurs early and may even be present before any change is noted in the primary lesion. On the other hand metastatic deposits may not appear until many years after the removal of the primary growth (Figs 926-927).

The outlook in melanocarcinoma is always poor only the exceptional patient making a recovery. In the presence of metastatic deposits life is usually measured in months.

The *benign melanoma* which may eventuate in melanocarcinoma should be dealt with circumspectly. The indications for the removal of such benign nevi have already been given (p. 3204). When melanocarcinoma is already present a combination of *radical surgery* and *roentgen therapy* should be used. Radiation therapy alone is inadequate since these neoplasms are not especially radiosensitive.

SARCOMA

Sarcomas are *malignant growths* which involve the nonepithelial structures of the skin. They are rare and are usually diagnosed by the pathologist rather than the clinician.

Skin sarcomas appear commonly as one or more raised or subcutaneous *nodules* with some discoloration of the skin. They are classified according to their histology into round cell or spindle cell sarcomas, fibrosarcomas, angiosarcomas and neurogenic sarcomas, the last being a form of degeneration occurring in the tumors of multiple neurofibromatosis (von Recklinghausen's disease). Melanosarcoma is an extremely rare malignancy due to sarcomatous metaplasia of a blue nevus (p. 3204).

The *treatment of cutaneous sarcomas* depends upon the variety of lesion present. Melanosarcoma should be dealt with in a manner similar to melanocarcinoma (p. 3225). The round cell sarcoma, spindle cell sarcoma and fibrosarcoma should be excised widely and roentgen rays or radium applied.

Neurogenic sarcoma is usually the result of surgical interference in neurofibromatosis. It is of a very low order of malignancy and may usually be left untreated.

IDIOPATHIC MULTIPLE HEMORRHAGIC SARCOMA (KAPOSI'S DISEASE)

Idiopathic multiple hemorrhagic sarcoma is a not unusual affection appearing most often in immigrants from Italy, Russia and Poland. It is most frequent in males past the age of forty-five years and occurs in patients in the lower economic strata.

The lesions usually appear first on the lower extremities about the foot and ankles, although early lesions may be seen elsewhere on the skin surface even in the mouth. The characteristic eruption consists of a group of multiple rounded nodules or flat plaques of a distinctive purplish brown or deep bronze color. The lesions are firm, generally nontender and smooth.

treatment of the local condition is purely protective (p 3136) the therapeutic program attempts to correct the fundamental disturbance

MERALGIA PARESTHETICA

Meralgia paresthetica is characterized by tingling numbness pain and formication in the *anterolateral aspect of the thigh*. There may be some objective sensory disturbance but the skin is usually unchanged in appearance

The cause of the affliction is pressure upon the *lateral femoral cutaneous nerve*. If the disturbance is not due to some obvious cause such as an ill fitting corset the fascia may be loosened *urgically*

CAUSALGIA

Causalgia is an extremely painful condition in the area supplied by an injured peripheral nerve. It is associated with the cutaneous alterations characteristic of neuritic atrophy of the skin (p 3078)



Fig 981.—P forating ulcers

Relief is afforded by well planned *alcohol* or *procaine* injections (p 2814) Symptomatic relief may be afforded by the application of *warm wet dressings*

LOCALIZED NEURODERMATITIS (LICHEN SIMPLEX CHRONICUS)

Lichen simplex chronicus is a localized skin condition which begins with severe intermittent pruritus and is followed by characteristic changes in the skin. The disease is far more frequent in the *female* and usually appears between the *fortieth* and *fiftieth* year. No exact causation is known but it is possible that the disease originates as a functional disturbance of a sensory nerve accompanied by itching and that the cutaneous changes are entirely secondary the result of scratching

This form of neurodermatitis has no relationship to the so called generalized neurodermatitis which represents one of the phases of atopic dermatitis

Clinical Manifestations—Usually only one or two patches of eruption are present the favored sites being the *nucha* the *sides of the neck* and the *extremities*. The individual patch varies from 2 to 3 cm to 10 to 15 cm diameter and is rounded or irregular in outline. It is slightly ele

ances are more frequent in the realm of the involuntary nervous system and include such familiar cutaneous manifestations of severe emotional stress as the blush of shame the pallor of fear the hyperidrosis and gooseflesh of strain and apprehension

The Skin and the Psyche—The skin enjoys a reciprocal relationship with the mind and emotions. Certain of the dermatoses are the result of psychogenic disturbances in which the skin is the *affective organ*. The recognition of these states is important since treatment lies rather in the realm of *psychiatry* than dermatology. Reversing the path of causal relationship certain of the dermatoses influence profoundly the mental state of the patient. Intractable itching especially in the vulva and anal regions may induce a state of marked *depression*. The uglier and more distasteful afflictions of the skin such as *acne vulgaris* may alter the entire *personality* *panel* of the sensitive individual.

Classification of Neurogenic Dermatoses—Mindful of the general principles which pertain to the interrelationship between skin and nervous system the discussion that follows is presented in the following categories

- Organic disturbances of the peripheral nerves
- Cutaneous disturbances of the involuntary nervous system
- Dermatoses of psychogenic origin
- Psychic effect of cutaneous disease

ORGANIC DISTURBANCES OF THE PERIPHERAL NERVES

The organic disturbances of the peripheral nerves include neuritic atrophy of the skin meralgia paresthetica causalgia localized neurodermatitis and the complicated conditions causing pruritus discussed at greater length elsewhere (p 3170)

NEURITIC ATROPHY OF THE SKIN

The area supplied by a nerve which has suffered injury or been damaged by disease presents a characteristic *glossy* appearance. This manifestation may follow *traumatic section* of the nerve *disuse* when an extremity has been immobilized lesions of the *peripheral nerve* such as occur in leprosy and diabetes mellitus and disturbances involving the *spinal cord* as seen in syringomyelia.

The appearance of the atrophic skin is smooth and shiny. The furrows are shallower than normal and may be completely obliterated. The hair is lost and trophic disturbances are seen in the nails. The color of the atrophic skin may be dead white but occasionally there is redness and blotchiness due to a local cutaneous dilatation of the small vessels.

PERFORATING ULCERS

With loss of the deeper pain and pressure sensations such as occur in leprosy diabetes mellitus tabes dorsalis and other cord lesions *perforating ulcers* are prone to occur particularly on the plantar aspect of the metatarsophalangeal junction of the great toe. These *trophic ulcers* are usually not large. Usually there is a sinus tract which extends to the bone. The area is anesthetic and the condition is painless.

The recognition of the trophoderm always calls for a complete physical and neurologic examination and the necessary laboratory procedures. The

applications are useful *Estrogenic therapy* may be cautiously attempted (p 2516)

PROGRESSIVE FACIAL HEMIATROPHY

Progressive facial hemiatrophy is a rare condition probably due to some pathologic process in the involuntary nervous system

The disturbance is characterized by progressive atrophy of the bones cartilages and soft parts of one half of the face leading to distinct *facial asymmetry* It usually progresses slowly to its maximum and arrests itself permanently It may be accompanied by evidences of a *Horner's syndrome* and any trauma which damages the *cervical sympathetic ganglia* may produce this condition At times it is seen in cases of *scleroderma*

Apparently *treatment* is ineffective

DERMATOSES OF PSYCHOGENIC ORIGIN

Physicians have long recognized the fact that profound disturbances of the *psyche* may give rise to *cutaneous manifestations* In *anxiety states* and *compulsion neuroses* (p 1357) somatic symptoms may predominate and involve the skin as well as other organs *Psychosomatic conversions* (p 1344) are useful to the disturbed patient who finds them a satisfying escape mechanism from the tortuous and insoluble conflicts that beset him mentally

Psychosomatic Disturbances.—The field of psychosomatic medicine is ever broadening and the future may reveal additional evidences of the significance of the mental states in cutaneous disease The human being must be regarded as a *total personality* (p 1359) in whom the character of any given disease is the result of an interplay of various forces rather than of a single stimulus True the patient is affected by the disease but conversely the disease follows a course conditioned by the esoteric variants of the individual's heredity his organic and functional peculiarities and his psychic make-up

The dermatoses that may be of *unquestioned psychogenic origin* include neurotic excoriations feigned eruptions trichotillomania dermatophobias urticaria and angioneurotic edema *Emotional overplay* is a contributing factor in the allergic dermatoses lichenification essential pruritus the cutaneous manifestations of disturbances of the involuntary nervous system rosacea and abnormalities in sweating

NEUROTIC EXCORIATIONS

Neurotic excoriations are *self induced lesions* produced by picking at the skin with the fingernails The patient suffers from a compelling urge to scratch or pick at the skin The eruption is most frequently seen in *females* from the adolescent period onwards No primary skin disease is present and all changes are caused by digital trauma The condition is considered a form of *compulsion neurosis* in which the patient suffers from "skin unrest" interpreted as sensations of itching or burning

Clinical Manifestations.—The lesions of neurotic excoriation are multiple superficial and crusted with a small amount of surrounding erythema They are usually small and fairly uniform in size and appearance They may heal without residuum but at times scarring and pigmentation may result

vated grayish or brownish and consists of closely crowded *papules*. These papules are flattened on top, dull and polygonal and the picture closely resembles the tessellation of a mosaic. Scaling is slight and fine. The pattern is that induced in the skin by prolonged scratching (*lichenification*) (p 3168).

Diagnosis—Lichen simplex must be differentiated from *lichen planus*, *chronic dermatitis venenata* and *psoriasis*. It lacks the discrete lesions, the violaceous hue, the characteristic distribution of the buccal lesions of *lichen planus*. The absence of vesicles at some time in the course of the disease militates against *dermatitis venenata*. *Psoriasis* usually is located in other areas and possesses a typical micaceous scale. *Histopathology* readily distinguishes between these various conditions.

Treatment—The most effective remedies are ointments of *tar* (p 3131) and *roentgen therapy*. *Crude coal tar* 1 to 5 per cent in petrolatum or in a paste of *zinc oxide* may be used. *Chrysarobin* 0.5 to 3 per cent in petrolatum may be effective. In stubborn cases painting the lesion with pure *liquid coal tar*, dusting with *talcum* and then applying a dressing may give excellent results. The antihistamine agent *pyribenzamine* merits trial (p 56).

Roentgen treatment produces rapid clearing of the lesions in most cases but recurrence is frequent. Underlying mental and endocrine disturbances should be sought and corrected whenever possible.

DISTURBANCES IN THE REALM OF THE INVOLUNTARY NERVOUS SYSTEM

Of dermatologic phenomena referable to disturbances in the realm of the involuntary nervous system only *acroparesthesia* and progressive facial hemiatrophy are described in the present section. *Urticaria* (p 3345) and *angioneurotic edema* (p 3349) are more often due to allergy than to psychogenic and neurogenic causation. *Erythromelalgia*, *Raynaud's disease* and *hereditary lymphedema of Milroy* reveal themselves as *angioneuroses* and accordingly they are described in the section devoted to *Circulatory Diseases* (p 1002).

ACROPAESTHESIA

Acroparesthesia is a *vasomotor neurosis* occurring chiefly in women after a natural or artificial menopause. Prolonged immersion of the hands in water seems to be a contributory factor. The subjective complaints include *tingling sensations*, *formication*, *numbness* and a sense of *tightness* of the hands. The feet are only rarely affected. The symptoms are at their worst at *night* and in the *morning* after awakening from sleep. Occasionally similar milder sensations are present in the tongue and lips.

The skin is cold, pallid and clammy but is otherwise normal. There may be such sensory disturbances as *hyperesthesia* or *hypesthesia*.

The etiology of the condition is unknown but it is believed to be the result of a *vasomotor sensory neurosis* precipitated by *endocrine dysfunction*. Some investigators regard the syndrome as a mild form of *Raynaud's disease* (p 1000).

Treatment—The patient is instructed to keep well covered in cold weather, to avoid the use of cold water on the body and to chew excesses of fatigue and prolonged immersion in water. Gentle massage and warm

(acids alkalis cantharides croton oil and so on) (2) mechanical devices or (3) by heat or cold

Clinical Manifestations—The feigned eruption may consist of *erythema vesicles bullae* or *necrosis* with *ulceration* dependent upon the concentration and duration of the action of the causative agent. The lesions are generally located in an area accessible to the hands and are frequently unilateral grouped bizarre and unnatural in appearance. Geometric patterns rarely observed in spontaneous dermatoses may be produced. Char-



Fig. 933.—Dermatitis artefacta. Self-inflicted lesions produced by the application of phenol. Notice that they are bizarre clean-cut and have a tendency to be linear where drops have run down the skin. (Courtesy of Dr. S. A. Oleynick.)

acteristic is the fact that new lesions keep appearing continually with remarkable suddenness.

Diagnosis—The diagnosis of the feigned eruption may not be easy but suspicion is aroused by the unnatural appearance and configuration, the mental state of the patient and the failure of the condition to fit into the picture of any known dermatosis. The discovery of the material used to cause the lesions and prompt healing when an occlusive dressing is used confirms the diagnosis. When a fixed dressing is placed over the eruption

In young females the eruption may be confined largely to the face and bears a superficial resemblance to *acne vulgaris*. However widespread distribution is common with involvement of the face, extremities and trunk.

Diagnosis—The absence of any primary skin lesion and the presence of uniform *crusted excoriations* on parts accessible to the fingernail indicate the correct diagnosis. *Secondary excoriations* are seen in many itching dermatoses (p 3178) and it is necessary to exclude such conditions as *pediculosis scabies pruritus* (essential or symptomatic) *urticaria* and *drug eruptions*. However the neurotic character of the patient is strongly suggestive the practitioner must remember therefore that the neurotic individual can also suffer from scabies, *urticaria* and other skin diseases.

Treatment—The patient is told that the skin lesions are the result of self inflicted trauma and that cure can only be obtained by complete

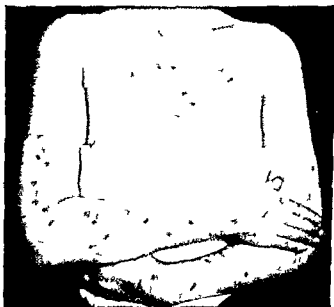


Fig 93°—Neurotic excoriations. Patient claimed to have been bitten by a flea five years before and ever since had been assiduously picking dead fleas out of the skin.

abstinence from picking and digging at the skin. Soothing antipruritic lotions or pastes may be of some value but in severe examples when the condition progresses psychiatric treatment is required.

FEIGNED ERUPTIONS (DERMATITIS FACTITIA, DERMATITIS ARTIFICIALIS)

A feigned eruption is a cutaneous lesion *artificially and purposefully produced* in an effort to obtain the interest and sympathy of others or to malingering.

Etiology—This unusual dermatosis is encountered most often in young hysterical females but has been described in criminals, military personnel, military draftees and industrial workers. In young females it is a manifestation of *hysteria* (p 1353) but, in the others it is usually employed as a means of evading duties, service or gaining undeserved compensation. The eruption is artificially induced most commonly by the use of (1) chemicals

CHAPTER 146

CUTANEOUS MANIFESTATIONS OF METABOLIC AND MISCELLANEOUS SYSTEMIC DISORDERS

MANY common skin conditions represent local manifestations of a wide spread metabolic disorder. The importance of recognizing these dermatoses and their interrelationships has two fold significance. Primarily the diagnosis assists in calling attention to the more profound abnormalities. Secondly from the standpoint of practical therapeutics it is obvious that treatment must be directed not at the local cutaneous manifestations but at the underlying systemic disturbance.

CUTANEOUS MANIFESTATIONS OF VITAMIN DEFICIENCIES

The vitamins are *essential substances* which cannot be elaborated by the tissues and must be *exogenously introduced*. Vitamin deficiencies most often arise from an *inadequate supply* in the dietary. *Improper eating* may be due to poverty, ignorance, food faddism, weight reduction diets, the anorexia of chronic disease, fever and severe acute illness or chronic alcoholism.

Less often the avitaminosis occurs despite an adequate intake when *abnormalities of alimentation, storage and excretion* are present. *Absorption* from the gastro intestinal tract may be impaired by diseases which induce persistent vomiting or diarrhea. Disease of the liver may interfere with the *conversion of carotin* (provitamin A) into vitamin A as well as with the *storage and formation of vitamin K*. In conditions associated with *increased need* for vitamins such as fevers and hyperthyroidism or with *increased elimination* as in diabetes mellitus or diabetes insipidus surplus amounts must be given in the diet to ward off deficiency. Prolonged ingestion of *mineral oil* may lead to vitamin A deficiency since the oil dissolves the vitamin or provitamin and eliminates it with the waste.

The general discussion of the vitamins and a description of clinical manifestations of avitaminoses other than those reflected in the skin are included in the section on *Metabolism* (p. 581). The cutaneous manifestations of avitaminoses and their diagnostic features are summarized in Table 160.

TABLE 160—CUTANEOUS MANIFESTATIONS OF AVITAMINOSES AND THEIR DIAGNOSTIC FEATURES

Vitamin A

Dryness (xerophthalmia) and ulceration of the cornea. Night blindness (p. 619). Dryness of the skin (xerosis). Spiny papules at mouths of hair follicles (keratosis pilaris).

Predisposition to pyoderms (p. 3248).

Note therapeutic response to large doses of Vitamin A. May be of value also in keratosis follicularis (p. 3296) and pityriasis rubra pilaris (p. 341).

Thiamine

Cutaneous atrophy secondary to polyneuritis (p. 1499). Edema in wet phase of beri-beri (p. 623).

Note therapeutic response to thiamine chloride. Try also in acrodynia (p. 3148).

new lesions cease appearing there although they may now break out in other uncovered skin regions

Treatment—Once the diagnosis is made treatment becomes simple and obvious The use of an *occlusive dressing* of glycerogelatin or plaster of paris and bland applications will lead to prompt healing However psychiatric treatment is advisable in most instances

TRICHOTILLOMANIA

Trichotillomania the compulsion to *extract hairs* is most often observed in individuals who are suffering from mental disturbances of a profound nature It is true that instances have been reported in persons who were apparently normal but such examples are distinctly in the minority

Adults are most often afflicted The *scalp* usually shows one area of incomplete *alopecia* which the patient has produced by pulling out and breaking off the hairs Otherwise the head is entirely normal and the residual hairs are not diseased

The majority if not all of these individuals are suffering from some *obsession* or a *profound psychiatric disturbance* and treatment lies in the realm of the *neuropsychiatrist* (p 1283)

DERMATOPHOBIAS

There is no doubt that the *phobic states* are symptoms of *mental disorder* and should be excluded from dermatologic therapy The dermatologist may encounter chiefly (1) the *bacteriophobe* the patient who is in dire fear of germs and shrinks from contact with human and inanimate objects washing his hands religiously after every inadvertent contact (2) the *syphilophobe* who catches syphilis from the most impossible sources (3) the *acarophobe* who is infested with lice and can reveal them readily by digging out a piece of skin and (4) the *cancerophobe* whose every skin mark is malignancy Whatever the manifestation these patients have a *psychiatric problem* and their treatment requires the attention of the specialist (p 1328)

VASCULAR LESIONS OF THE SKIN

The vascular lesions of the skin do not form a homogeneous group For the most part they are symptomatic and are elsewhere considered They include

Neoplasms

Angiomas (p 3200)

Lymphangiomas (p 3203)

Nevi (p 3204)

Telangiectases (p 3394)

Hemorrhages

Ecchymoses (p 3122)

Purpuras (p 3122)

Petechiae (p 3398)

Alterations in the Color of the Lume

Erythemas (p 3162)

Angiospasm (p 1000)

CHAPTER 146

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MANY common skin conditions represent local manifestations of a wide spread metabolic disorder. The importance of recognizing these dermatoses and their interrelationships has two fold significance. Primarily the diagnosis assists in calling attention to the more profound abnormalities. Secondly from the standpoint of practical therapeutics it is obvious that treatment must be directed not at the local cutaneous manifestations but at the underlying systemic disturbance.

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Note their peculiar response to large doses of Vitamin A. May be of value also in keratosis follicularis (p 3236) and pityriasis rubra pilaris (p 3112).

Thiamine chloride

Cutaneous atrophy secondary to polyneuritis (p 1499). Edema in "wet phase" of beriberi (p 625).

Note therapeutic response to thiamine chloride. Try also in acrodynia (p 3148).



Fig 934 —Follicular hyperkeratosis of vitamin A deficiency *



Fig 936 —Rickets due to vitamin D deficiency *



Fig 935 —Hemorrhagic jaundice of vitamin K deficiency *



Fig 937.—Pitting edema of legs ("wet beri beri") and peripheral neuritis of vitamin B deficiency *



Fig 938—Photophobia epiphora and scleral injection in riboflavin deficiency



Fig 939—Cheilitis and photophobia in riboflavin deficiency



Fig 940—Glossitis of niacin deficiency

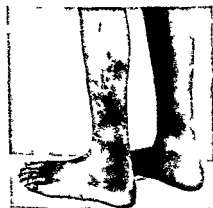


Fig 941—Pellagrous dermatitis of niacin deficiency



Fig 942—Pellagrous dermatitis of hands in niacin deficiency

Riboflavin

Dryness and scaliness of skin. Fissures and chapping of vermillion surfaces of lips (cheilosis). Red greasy eruption with fine follicular projections of nasolabial folds. Fissures at commissures of mouth. Magenta tongue with flattening of papillae (p 1674). Superficial vascularizing keratitis (p 1676).

Note therapeutic response to riboflavin. Try also in acne rosacea (p 3357).

Niacin

Pellagra with mental neurogenic and digestive manifestations. Diffuse redness, edema, desquamation, hyperpigmentation, vesiculation and bulla formation of exposed surfaces. Redness and erosion of tongue with flattening of papillae (p 3237).

Note therapeutic response to nicotinic acid.

Biotin

Due to binding of biotin by avidin in egg white. Productive of generalized exfoliative dermatitis.

Note therapeutic response to diet free from egg white.

Para aminobenzoic acid and pantothenic acid

Loss of hair color (achromotrichia) in experimental animals. Administration of 100 mg of para aminobenzoic acid twice daily for restoration of color to gray hair not impressive.

Vitamin C

Scurvy with cutaneous hemorrhages, petechiae, ecchymoses and purpura. Usually associated with bleeding gums.

Note diminished resistance of capillaries as revealed by tourniquet test. Try therapeutic response to ascorbic acid (p 627).

Vitamin K

Cutaneous hemorrhages in hemorrhagic disease of newborn (p 2749). Ecchymoses in deficiencies associated with jaundice (p 1951).

Note therapeutic response to menadione (p 630).

CUTANEOUS MANIFESTATIONS OF THE ENDOCRINOPATHIES

The cutaneous manifestations seen in the endocrinopathies are mostly of *diagnostic importance* calling to the attention of the practitioner the more fundamental metabolic disorder. Abnormalities of the skin occur both in hyper- and hypofunctioning of the various glands. Under the latter circumstance the dermatosis is corrected by *substitution therapy*.

TABLE 161.—CUTANEOUS MANIFESTATIONS OF THE ENDOCRINOPATHIES AND THEIR DIAGNOSTIC FEATURES

Hyperthyroidism

Skin delicate, transparent, warm and moist. Hyperidrosis, dermatographia, pruritus, urticaria, flushing, blushing, cold hands and feet, chilblains, premature graying of hair, alopecia and trophic changes of nails (p 1197). Other patients frequently develop hyperpigmentation but there is no involvement of the mucous membranes as in Addison's disease (p 1271).

Note therapeutic responses to iodid, Dercil and subtotal thyroidectomy (p 1210).

Hypothyroidism

Cretinism and myxedema with thickened puffy skin which does not pit on pressure. Eyelids appear baggy. Skin dry and roughened. Sweat secretion diminished or absent. Delicate maler flush. Hair scant. Outer third of eyebrow missing. Nails brittle and easily broken.

Note therapeutic response to thyroid extract (p 1189).

Hypoparathyroidism

Chronic tetany (p 1253) associated with alopecia, puffiness of skin, trophic changes in nails and the formation of cataracts (p 1592). Werner's (Rothmund) syndrome, a rare hereditary and familial disturbance with premature baldness and graying of the



Fig 913—Gingivitis of scurvy in vitamin C deficiency. Angular scarring of lips from associated riboflavin deficiency.



Fig 914—Capillary hemorrhages in adult scurvy due to vitamin C deficiency.
Courtesy of Eli Lilly and Co.

hair precocious cataract formation hypogonadism osteoporosis metastatic calcinosis
scleroderma and trophic ulcers of legs and ankles
Try parathyroid hormone (p 1223)

Hyperparathyroidism

Metastatic calcinosis

Consider parathyroidectomy (p 1231)

Adrenal cortical deficiency

Addison's disease with pigmentary changes of exposed surfaces and mucous membranes due to increased melanin content

Note response to sodium chloride and cortical extracts (p 1267)

Hyperadrenalemia

Precocious puberty with hirsutism acne vulgaris and hypertrichosis (p 1182)

Look for masculinizing tumor (p 2481)

Hyperpituitarism

Thickening and roughness of skin in acromegaly (p 1156) Hair thick and abundant

Yellow to brown areas of pigmentation on face neck and chest

X ray sella turcica Examine fundi and plot visual fields

Hypopituitarism

Obesity with thin skin and sparse hair in adiposogenital syndrome (Frohlich) Atrophy and pigmentation of skin in cachexia hypophyseopriva (Simmonds) Masculinization obesity hirsutism and cutaneous striae in pituitary basophilism (Cushing) (p 1159)

Hypo insulinemia

Diabetes mellitus with generalized itching (p 1216) Tendency to develop pyoderms

Carotinemia with lemon yellow pigmentation with vitamin A deficiency Xanthomas of extensor surfaces of forearms elbows knees and back Yellowish papules or linear discolorations also appear in these areas as well as on palms and soles (p 1218)

Necrobiosis lipoidica diabetorum consists of nodular infiltrations of anterior aspects of thighs where insulin has been injected Trophic changes secondary to peripheral neuropathy and associated arteriosclerosis of smaller vessels

Note effects of dietary control and insulin (p 1255)

Gonadal disturbances

Menstruation

Erythema pruritus herpes simplex urticaria and aggravation of acne vulgaris (p 3328)

Pregnancy

Hyperpigmentation of face (chloasma) nipples areolae abdominal wall and genitals

Linea nigra Abdominal striae Herpes gestationis in successive pregnancies a benign variant of dermatitis herpetiformis (Fig 915) Impetigo herpetiformis with grouped pustular lesions which coalesce may be associated with fever chills and delirium requiring interruption of pregnancy (Fig 916)

Adolescence

Acne vulgaris probably due to dislocation of estrogen androgen ratio (p 227)

Menopause

Keratoderma climactericum with hyperkeratoses and fissures of palms and soles

Leukoplakia vulvae Kraurosis vulvae (p 2397) Pruritus vulvae (p 2591)

Administer estrogen with great caution (p 217)

Senility

Atrophy of skin Keratoses and pigmentation Senile vulvovaginitis (p 322) Senile pruritus (p 314)

Administer estrogen or androgen with great caution

CUTANEOUS MANIFESTATIONS OF METABOLIC DISORDERS

Changes in the skin are observed in many metabolic disorders such as aberrations in the chemistry of lipids uric acid calcium iron and amyloid They are listed in Table 167

Xanthelasma Palpebrarum—Xanthelasma palpebrarum is an eruption confined to the *eyelids*. The lesions are smooth soft slightly elevated vel lowish *papules* varying in size from 1 to 25 mm. They occur most commonly on the upper lids near the inner canthus but are not infrequently present on the lower lids. They are somewhat commoner in *females* and appear usually after the age of thirty years. They increase in size very slowly reaching a point of maximum development when growth ceases. A family history of similar lesions is frequently present.



Fig 945—Dermatitis herpetiformis of pregnancy



Fig 946—Necrobiosis lipoidica diabetorum



Fig 947—Xanthelasma (eyelids)



Fig 948—Xanthoma tuberosum (hands)

The eruption represents a derangement of the cellular *lipid metabolism* but it is rarely accompanied by visceral manifestations or *hypercholesteremia*.

Treatment—Local treatment is most effective but *recurrences* are frequent. When the skin of the eyelids is redundant and the lesions are not excessively large *excision* may yield a good cosmetic result. *Desiccation* with a very fine electrical spark is frequently successful. A favorite procedure among dermatologists is the application of a chemical caustic preferably *trichloroacetic acid* (p 3131). The healthy skin about the lesion should be protected with a layer of petrolatum and the acid applied to the surface of the lesion with a glass rod or a pointed wooden stick. When the tissue becomes white no further acid should be applied but alcohol may be used to remove any excess acid. Several days after this treatment a

 DIFFERENTIAL DIAGNOSIS OF

Generalized Pigmentation

The normal color of the skin is dependent upon the quantity and quality of the blood in the capillary bed the thickness of the skin and the content of melanin an iron free pigment formed in the basal layer of the epidermis

See also Pigmented Dermatoses p 3154

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|--------------------------------|---|
| Actinic | From exposure to natural or artificial sunlight |
| Addison's disease | Brown to black pigmentation most marked over frictional areas Also noted on mucosal surfaces Note hypotension and asthenia Therapeutic response to sodium and adrenal cortical extract (p 1267) |
| Argyria | Slate grey pigmentation due to excessive use of silver salts internally or by urethral instillation |
| Arsenic | Brown pigmentation from prolonged arsenotherapy with inorganic preparations |
| Atabrine | Yellow pigmentation sparing cornea due to use of antimalarial Not accompanied by disturbances of bile pigments as revealed by urine stool and serum examinations |
| Cachexia | Yellowish discoloration of skin in malignancy chronic malaria subacute bacterial endocarditis uremia and other chronic diseases Make complete physical examination |
| Carotinemia | Yellow pigmentation most marked in palms and soles Due to excess of provitamin A Unassociated with jaundice as revealed by examinations of urine stool and serum |
| Chrysisis | Tan to brown pigmentation due to therapeutic use of gold |
| Hemochromatosis | Presence of iron containing hemosiderin in skin Associated with glycosuria hepatomegaly and atrophy of testicles Bronzed diabetes (p 1976) |
| Hyperthyroidism | Brown pigmentation Associated with tachycardia and elevation of B M R Note therapeutic response to iodide and Deracil |
| Jaundice | Yellow to greenish pigmentation involving cornea as well as skin Note disturbances of bile pigment metabolism as revealed by urine stool and serum examinations (p 1951) |
| Macrocytic hyperchromic anemia | Pernicious anemia with yellow pigmentation of skin but clear cornea Bile pigments not demonstrable in urine or serum Hemogram reveals high color index and increased size of individual erythrocytes Note therapeutic response to liver extract (p. 1077) |

| | |
|-------------|--|
| Ochronosis | Blue discoloration of cartilages of ear Dark discoloration of urine probably dependent on disordered metabolism of phenol or one of its homologues especially tyrosine May be associated with fragilitas osseum and pathologic fractures of bone Examine urine for evidences of alkaptonuria |
| Sensibility | Yellowish discoloration of skin which also reveals lack of elasticity wrinkling atrophy and areas of pigmentation and keratosis |

crust forms and boric acid ointment may be used The acid treatments may be performed at two or three week intervals until the lesion has been brought down to normal skin level at which time the yellowish color is usually dissipated

TABLE 102—CUTANEOUS MANIFESTATIONS OF METABOLIC DISORDERS AND THEIR DIAGNOSTIC FEATURES

| | |
|----------------------------|--|
| Diabetes mellitus | See p 1216 |
| Gout | Tophi of ear fingers and toes Identify sodium urate crystals |
| Ret iculo endoth el i osis | Xanthomas and pinguiculae in Gaucher's and Niemann Pick syndromes |
| Hemochromatosis | Bronzing of skin due to deposits of iron-containing hemosiderin Associated with diabetes mellitus cirrhosis of liver and pancreas and testicular atrophy |
| Ochronosis | Familial and hereditary metabolic disorder Alkaptonuria with homogentisic acid in urine Blue discoloration of cartilages of nose and ears Grey to black deposits of sclerae |
| Calcinosis | |
| Local red | Calcinosis circumscripta with isolated radiopaque deposits about small joints of hands and extensor surfaces of larger joints May be associated with scleroderma or Raynaud's disease May be metastatic in conditions associated with bone destruction (metastatic malignancy hyperparathyroidism or multiple myeloma) |
| Generalized | Calcinosis universalis As localized variety but with more extensive deposits on contractures and ankyloses Usually fatal |
| Amyloidosis | |
| Lichen amyloidosis | A cutaneous disorder with tan or brown papules and nodules Intense pruritus Local injection of 1 cc of 1.5% Congo red dyes lesions red or pink Try x-ray treatment |
| Generalized amyloidosis | Systemic disorder with hepatosplenomegaly Usually secondary to chronic suppuration |
| Systematized amyloidosis | Smooth skin papules and plaques at mucocutaneous junction Biopsy reveals amyloid infiltrate of corium Associated deposits in skeletal and heart muscles cause diffuse aches and pains macroglossia and myocardial disturbances Usually fatal |

In the presence of hypercholesteremia (p 736) a low fat diet (p 697) is indicated

Xanthoma Tuberosum et Planum—Xanthoma tuberosum et planum is an eruption of yellow or orange papules and nodules These lesions are most often situated on the extensor surface of the arm the elbow and on the

buttocks While they do occur in other locations they never are seen in the axilla or the elbow or knee bends which is an important differential point from xanthoma disseminatum Linear yellowish xanthomatous deposits appear in the natural creases of the palms and digits The papules and nodules are firm and nontender and frequently have a *hyperkeratotic* surface

In this condition there is almost invariably an elevation of the *blood cholesterol* (p 736) and a frequent association with *visceral lesions* There may be *tendon xanthoma* which appears as subcutaneous nodules attached firmly to the tendons and ligaments especially the Achilles and digital tendons These are firm painless nodules varying in size from a pea to a small egg The *intima of the blood vessels* especially the *coronary arteries* and the *endocardium* may be affected A type of *biliary cirrhosis* due to deposits in the ductal system has been described

Xanthoma Disseminatum—Xanthoma disseminatum consists of slightly elevated *nodules* or *papules* at first yellowish and later dark brown in color They are small lesions but cluster together closely to form *plaque like* masses They have a predilection for the axillae the lateral aspects of the neck and the bends of the elbows and knees Similar lesions may be present in the mucosa of the mouth the epiglottis the larynx the cornea and sclera The eruption does not itch The *blood cholesterol* is within normal limits

Xanthoma disseminatum may occur only in the skin and mucosa or it may be accompanied by important visceral changes It may be seen with *diabetes insipidus* and evidences of brain involvement Deposits may be present in the *bones* particularly the skull pelvis scapula and long bones Lung involvement has been reported

Schuller Christian's syndrome is often associated with xanthoma disseminatum of the skin and mucous membranes The distinctive symptoms (defects of the skull bones exophthalmos and diabetes insipidus) are due to xanthomatous deposits

CHAPTER 147

THE SKIN IN INFECTION PORTALS OF ENTRY PRIMARY AND SECONDARY MANIFESTATIONS

| | |
|--|------------------------------|
| Staphylococcus Infection | Typhus Fever |
| Streptococcus Infection | Rocky Mountain Spotted Fever |
| Conococcus Infection | Orova Fever |
| Meningococcus Infection | Trench Fever |
| Pneumococcus Infection | Taatsugamushi Fever |
| Typhoid and Paratyphoid Bacilli Infections | Upper Respiratory Infections |
| Infection with the Tubercle Bacillus | Acute Mononucleosis |
| Diphtheritic Infection | Rubella |
| Leprosy | Measles |
| Anthrax | Erythema Infectiosum |
| Brucellosis | Roseola Infantum |
| Plague | Chickenpox |
| Tularemia | Smallpox |
| Glanders (Farcy) | Alastrim |
| Erysipeloid | Generalized Vaccinia |
| Clancroid | Molluscum Contagiosum |
| Syphilis | Warts |
| Framboesia Tropica (Yaws) | Herpes Simplex |
| Pinta | Herpes Zoster |
| Erosive and Gangrenous Balanitis | Epidemic Encephalitis |
| Relapsing Fever | Lymphopathia Venereum |
| Infectious Jaundice | Granuloma Inguinal |
| Rat Bit Fever | Dengue Fever |
| Haverhill Fever | Yellow Fever |
| | Foot and Mouth Disease |

The tegumentary barrier is the first and most powerful of the defenses of the body against invasion by living parasites. It is more than likely that pathogens attack the host only when the integrity of the dermal structures has been compromised.

The cutaneous reaction to infection is a variable. The integument may act as a *portal of entry* the sites of the inoculum presenting no characteristic features. There may develop at the site of penetration a characteristic *initial lesion* the recognition of which is of importance to the practitioner since at this time prompt and efficient local and systemic therapy usually offers the best chance for complete and permanent cure. Another cutaneous reaction to infection involves the role of the skin as a *mirror* for the pathologic process that results from the systemic invasion. By the characteristic *rash* the practitioner is enabled to diagnose the nature of the invading organism and institute adequate therapy.

The dermatologic aspects of infection are summarized in Table 163. The invading organisms are listed in the left hand column followed by notations on their primary and secondary manifestations. By referring to the right hand columns the reader can determine whether diagnosis is based on morphological appearance, culture of organism, or serologic or skin tests.

CARBUNCLE

A carbuncle is the result of the simultaneous infection by the *Staphylococcus aureus* of several adjacent hair follicles. This lesion is seen most frequently in men past middle age. Diabetes mellitus, malnutrition and anemia are predisposing factors.

The Eruption—The carbuncle is larger than the furuncle, at times reaching a diameter of 6 to 8 inches. It has *multiple orifices*. The lesion is slow in development and may require weeks to suppurate, evacuate and heal. There is deep as well as wide involvement of the skin. Redness, tenderness and induration are very marked. The favorite sites for the carbuncle are the *nape of the neck*, the *upper back* and the *buttocks*. *Systemic symptoms*, fever, chills and malaise are frequent and severe. (Fig. 949.)

Diagnosis—The diagnosis of the carbuncle is usually quite simple. The lesion may be confused with the malignant pustule of anthrax (p. 292). The latter is relatively rarely observed and is more apt to be associated with local gangrenous destruction and more grave systemic symptoms. *Bacterologic investigations* may be needed to make a certain diagnosis. A broken down gumma (p. 3286), a lesion of considerable rarity, may simulate the carbuncle. The syphilitic lesion is relatively painless and presents fewer inflammatory signs; toxemia is absent; the serologic test of the blood is often positive and the response to anti-syphilitic therapy is striking.

Treatment—See p. 3256.

PUSTULAR FOLLICULITIS (IMFETIGO OF BOCKHART)

Pustular folliculitis is characterized by the formation of superficial pustules at the follicular apertures. The causative agent is the *Staphylococcus aureus*. Men are most frequently affected though the eruption may occur in both sexes and at all ages. *Occupations* which are dirty and involve the handling of oils, greases, tars and certain chemicals predispose to the lesion. Outbreaks may follow the application of tar and other ointments to hairy regions. They occur too in regions subjected to scratching or those soiled by purulent discharges.

The Eruption—The pustule is superficial and situated exactly at the aperture of the *hair follicle*. Multiple lesions are present on the bearded region of the face, the neck and the extremities. (Fig. 949.)

Diagnosis—In the beard the condition must be differentiated from *syphilis vulgaris* (p. 3249) which is also a type of pustular folliculitis. *Iodides* and *bromides* (p. 3339) may produce similar lesions. *Furuncles* are deeper and exhibit more redness, elevation, pain and induration.

Treatment—See p. 3256.

SYCOSIS VULGARIS (SYCOSIS BARBAE)

Sycosis vulgaris is a chronic inflammatory disease of the hair follicles of the *male beard*, occasionally involving also the *eyebrows*, *axillae* and *pubis*. The infecting organism is the *Staphylococcus aureus*. The disease affects adult males almost exclusively. No predisposing or underlying factors are known; the disease frequently attacking men in perfect health.

Pathology—The essential pathology is a deep inflammatory and suppurative infection of the hair follicle and perifollicular tissues.

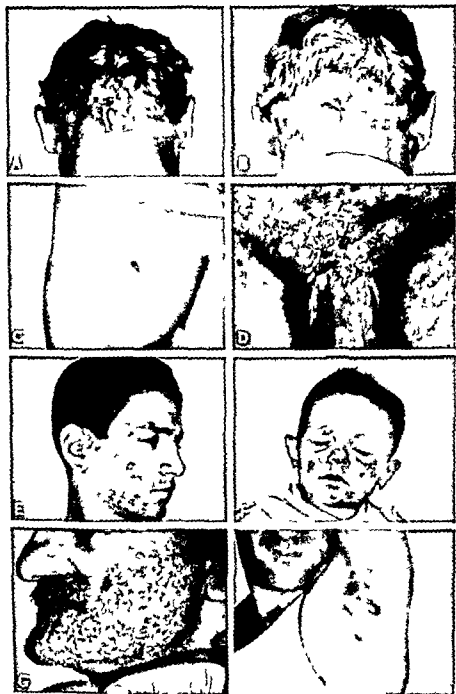


Fig 910—A Carbuncle before penicillin B Carbuncle after penicillin C Furuncle D Pustular folliculitis E and F Impetigo contagiosa G Syphilis vulgaris H Axillary sweat gland abscess

DIFFERENTIAL DIAGNOSIS OF

Painful Dermatoses

| | LESION | SITE OF PREDILECTION | REMARKS |
|-----------------------------------|--|---------------------------------|--|
| Acrodynia (p 3145) | Swelling and redness | Hands and feet | In infancy Try effects of vitamin B |
| Acroparesthesia (p 3230) | Pallor | Hands and feet | With tingling In females |
| Adiposis dolorosa (p 1164) | Tumors | None | Painful lipomas Excise if possible |
| Bites (p 3180) | Punctum | Exposed parts | Especially bees wasps spiders and reptiles |
| Causalgia (p 3229) | Atrophy | Course of injured sensory nerve | Treat by procaine and alcohol injection |
| Chilblain (p 3173) | Redness swelling and vesiculation | Fingers toes nose and ears | Due to frostbite Consider use of anticoagulants |
| Corn (p 3165) | Hyperkeratoses | Small toe Planar surfaces | Relieve pressure points Consider x ray treatment if keratolytics fail (p 3796) |
| Dermatitis herpetiformis (p 3241) | Grouped papules vesicles pustules and bullae | Bilateral and symmetrical | With pigmentation |
| Erythema nodosum (p 3375) | Bluish nodules | Extensor surfaces of legs | Due to hypersensitivity perhaps to bacterial protein. Isolate offending allergen if possible |
| Erythromelalgia (p 1002) | Redness and swelling | Extremities | Bilateral and symmetrical |
| Glomus tumor (p 3207) | Nodule | Often subungual | Excise |
| Herpes zoster (p 435) | Vesicles | Course of cutaneous nerve | Preceded by paresthesia and followed by neuralgia |
| Meralgia paresthetica (p 3223) | None | Anterolateral thigh | From pressure on cutaneous nerve |
| Pyoderma (p 3248) | Pustules | Nose ear hands and feet | Staphylococcal lesions Try local or systemic antibiotic therapy |
| Solar dermatitis (p 3174) | Sunburn | Exposed parts | |
| Verrucae (p 3288) | Warts | Plantar especially | Treat by excision or x ray |
| Vulvovaginitis (p 2587) | Fissures and atrophy | | Precancerous senile lesion |

Eruption—The involved area is red and swollen and contains many *pustules* and *papules*. Each lesion is located at the orifice of a *hair follicle* and is pierced by the hair. A certain amount of deep infiltration is present in the patches. The oozing and crusting may produce a picture resembling *eczema*. A solitary area may be involved on the upper lip or chin; there may be a number of separated discrete areas or the entire beard and moustache may be affected (Fig 949 E & F).

Diagnosis—*Sycosis barbae* must be differentiated from simple *pustular folliculitis* (p 3240) in which the lesions are more superficial and discrete and in which response to therapy is usually prompt. *Tinea infections* (p 3293) of the beard are localized but more inflammatory; deeper in extent and more inclined to abundant pus formation. *Bromides* and *iodides* (p 3339) occasionally produce a localized eruption resembling *sycosis vulgaris*.

Treatment—See p 3256.

IMPETIGO CONTAGIOSA (IMPETIGO VULGARIS)

Impetigo contagiosa is a contagious superficial infectious disease of the skin attended by the formation of *vesicles*, *bullae* and *crusts*.

Etiology—The causative organisms are the *Staphylococcus aureus* and *albus* and the *Streptococcus haemolyticus*. The disease is transmitted by direct contact with the fingers by kissing or the use of infected towels, brushes or razors. Men are likely to acquire the infection in barber shops. It is most frequent in children; young adults are not uncommonly affected.

The lesion—The earliest lesion is a spot of *erythema* which soon becomes *vesicular* or *bullous* and finally *pustular*. The lesions rupture, dry and form yellowish superficial (stuck on) *crusts*. Removal of the crust reveals a reddish moist eroded surface. There are practically always multiple lesions, autoinfection being the rule. The individual lesion is rounded or oval and may form rings which are clear centrally. The *exposed surfaces* of the face, ears, neck and hands are the favorite sites (Fig 949 E & F).

In children it is not rare to find an associated albuminuria and other evidences of a low grade *nephritis*.

Impetiginization—The term *impetiginization* refers to impetigo like lesions which are engrafted upon a skin area afflicted with some itching dermatosis. Such secondary impetigo is seen in infantile *eczema* and in *pediculosis capitis*.

Diagnosis—Differentiation from *herpes simplex* (p 433) of the face may be difficult. In *herpes* the lesions are less regular in form; there are vesicles and not bullae and there is not the same tendency to spread. In *tinea circinata* (p 3295) the central area shows some evidence of clearing; typical crust formation is absent; progress is less rapid and fungi can be demonstrated. In *impetiginized dermatoses* (*eczema*, *pediculosis*) there are definite evidences and a history of the pre-existing pruritic eruption. The lesions of a *bullous erythema multiforme* (p 3374) have a typical distribution on the lips, extensor surfaces of the extremities and the trunk. At times the buccal mucosa is involved and the eruption is more acute and widespread.

Clinical Variants—There are three special forms of impetigo which merit individual consideration

Furfuraceous Impetigo—Furfuraceous impetigo is encountered only in *children*. It consists of one or more rounded superficial pink dry scaly areas usually situated on the *cheeks*. The responsible organism is the *hemolytic streptococcus*

Bullous Impetigo Contagiosa (Pemphigus Neonatorum)—Bullous impetigo contagiosa is a more or less *generalized eruption of newborn infants* occurring in an isolated instance or appearing in *epidemic* form in hospitals nurseries or foundling homes. It is caused by the *Staphylococcus aureus* is distinctly contagious and may affect healthy infants

The *eruption* consists of few or very many bullae distributed generally over the body. These rupture and form crusted lesions. There may be universal involvement and a conversion of the cutaneous picture into a *generalized exfoliative dermatitis* or the disease may be mild and readily amenable to therapy. At times it is attended by severe constitutional symptoms with fatal outcome

Dermatitis Exfoliativa Neonatorum (Ritter's Disease)—Dermatitis exfoliativa neonatorum is believed to be a sequence of the bullous form of impetigo contagiosa. Exfoliation follows closely upon the bursting and drying of the vesicles and bullae. The skin generally is dry red and scaly but some vesiculobullous lesions may remain. Constitutional symptoms may be severe with high fever vomiting and dehydration

Treatment—See p 3256

ECTHYMA

Ecthyma is a *pyogenic* infection of the skin seen chiefly in individuals living under poor hygienic conditions or afflicted with wasting diseases. The infecting organism is usually the *Staphylococcus aureus* although *streptococci* are often present. Persons who live in dirty surroundings with inadequate bathing facilities and those suffering from lowered resistance due to chronic alcoholism or wasting diseases are mostly affected. The disease is most frequent in adult males. Itching diseases of the skin may act as a predisposing factor

The Eruption—The lesions consist of *pustules* which extend deeply quickly rupture and form a dark brown adherent *crust*. From the edges of this crust there is an exudation of purulent matter. When the crust is loosened and freed the base is seen to be a shallow *ulcer* which in time heals leaving a *pigmented scar*. Multiplicity of lesions is the rule. The *buttocks* and *legs* are the sites of predilection

Diagnosis—The eruption must be differentiated from *impetigo contagiosa* (p 3251) which is a much more superficial process and never produces a scar from *simple furuncle* (p 3248) which does not form crusts or ulcers from *psoriasis* (p 3414), which is dry and not crusted from the *rupial syphilide* which is seen most frequently on the face chest and arms has a stratified and dryer crust and is accompanied by positive serologic tests for syphilis from *varicose ulcers* which commonly affect the lower third of the leg and are associated with varicose veins edema and evidences of dermatitis hemostatica (varicose eczema) (p 3371)

Treatment—See p 3256

AXILLARY SWEAT GLAND ABSCESES (HIDRADENITIS SUPPURATIVA AXILLARIS)

Infection of the apocrine sweat glands (p 3501) of the axilla may be caused by the *Staphylococcus aureus*. The disease is more common in females though men are occasionally affected. It is most frequent in young adults and apparently does not occur before the age of puberty. Predisposing factors include excessive sweating, alterations in the chemical constitution of the sweat, shaving and the use of *deplatories* and *anidrotics*. The condition is seen more often in the *summer*.

The Lesion—One axilla may be affected, both may be involved simultaneously, or after the original area is well, the other axilla may later be

DIFFERENTIAL DIAGNOSIS OF

Dermatoses of the Axilla

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|--------------------------------|---|
| Acanthosis nigricans | Rare dermatosis characterized by pigmentation and papillary excrescences. Often associated with intra abdominal malignancy (p 3355). |
| Axillary sweat gland abscesses | Deep seated staphylococcal infections. Consider systemic penicillin therapy (p 106). |
| Contact dermatitis | Allergic dermatosis caused by articles of clothing, particularly dress shields. Remove offending allergen (p 3330). |
| Intertrigo | Dermatitis due to friction. Particularly in the obese with diffuse perspiration (p 3161). |
| Monilia | Yeast infection of intertriginous area. Identify pathogen (p 502). |
| Scabies | Burrows with intense itching. Occasionally found in axillae in addition to webs of fingers, wrists, genitals and abdomen. Identify pathogen (p 3180). |
| Seborrheal dermatitis | Dry scaly macular areas or moist plaques. Associated with seborrhea of the scalp. |
| Trichomycosis nodosa | Dystrophy of axillary hair. Note pin sized brown or yellow masses on shafts of hairs (p 3449). |

come diseased. The disease appears as a tender localized red *swelling* which bears close resemblance to a furuncle. There may be one or multiple lesions. After an interval the lesions soften, become fluctuant and incision yields thick purulent material. After evacuation healing commences but the area of induration may remain for many weeks. There is a tendency for lesions to appear in sequence. As one lesion is healing a fresh infection appears in a healthy area. (Fig 949 H)

Diagnosis—The diagnosis presents no problem except that the condition has been mistaken for furunculosis of the axilla and suppurative lymphadenitis. In *furunculosis* (p 3248) the lesions are more superficial, they progress more rapidly, have a yellow cap at the summit and are unusual in this location. *Suppurative lymphadenitis* (p 106) is a deeper process.

Clinical Variants—There are three special forms of impetigo which merit individual consideration

Furfuraceous Impetigo—Furfuraceous impetigo is encountered only in children. It consists of one or more rounded superficial pink dry scaling areas usually situated on the cheeks. The responsible organism is the hemolytic streptococcus.

Bullous Impetigo Contagiosa (Pemphigus Neonatorum)—Bullous impetigo contagiosa is a more or less generalized eruption of newborn infants occurring in an isolated instance or appearing in epidemic form in hospitals, nurseries or foundling homes. It is caused by the *Staphylococcus aureus* is distinctly contagious and may affect healthy infants.

The eruption consists of few or very many bullae distributed generally over the body. These rupture and form crusted lesions. There may be universal involvement and a conversion of the cutaneous picture into a generalized exfoliative dermatitis or the disease may be mild and readily amenable to therapy. At times it is attended by severe constitutional symptoms with fatal outcome.

Dermatitis Exfoliativa Neonatorum (Ritter's Disease)—Dermatitis exfoliativa neonatorum is believed to be a sequence of the bullous form of impetigo contagiosa. Exfoliation follows closely upon the bursting and drying of the vesicles and bullae. The skin generally is dry, red and scaly but some vesiculobullous lesions may remain. Constitutional symptoms may be severe with high fever, vomiting and dehydration.

Treatment—See p 3256

ECTHYMA

Ecthyma is a pyogenic infection of the skin seen chiefly in individuals living under poor hygienic conditions or afflicted with wasting diseases. The infecting organism is usually the *Staphylococcus aureus* although streptococci are often present. Persons who live in dirty surroundings with inadequate bathing facilities and those suffering from lowered resistance due to chronic alcoholism or wasting diseases are mostly affected. The disease is most frequent in adult males. Itching diseases of the skin may act as a predisposing factor.

The Eruption—The lesions consist of pustules which extend deeply, quickly rupture and form a dark brown adherent crust. From the edges of this crust there is an exudation of purulent matter. When the crust is loosened and freed the base is seen to be a shallow ulcer which in time heals leaving a pigmented scar. Multiplicity of lesions is the rule. The buttocks and legs are the sites of predilection.

Diagnosis—The eruption must be differentiated from impetigo contagiosa (p 3251) which is a much more superficial process and never produces a scar from simple furuncle (p 3248) which does not form crusts or ulcers from psoriasis (p 3414) which is dry and not crusted from the rupial syphilide which is seen most frequently on the face, chest and arms has a stratified and dryer crust and is accompanied by positive serologic tests for syphilis from varicose ulcers which commonly affect the lower third of the leg and are associated with varicose veins, edema and evidences of dermatitis hemostatica (varicose eczema) (p 3371).

Treatment—See p 3256

| | |
|----------------------|---|
| Seborrhea | Excessive oiliness or dryness of scalp often associated with seborrheal dermatitis Characterized by dry scaly macules or moist plaques of forehead cheeks or presternal regions |
| Syphilis | Pigmented lesion of leukoderma colli with whitening around neck Get serologic test (p 336) |
| Tinea capitis | Ringworm of scalp Note partial alopecia with broken off hairs Identify fungus by smear or culture (p 3293) |
| Trichorrhexis nodosa | Pare dystrophy of hairs Nodular swellings on shafts Splitting of hairs May be due to toilet ries (p 3449) |
| Canities | Graying of the hair Often familial |
| Trichotillomania | Compulsion to extract hairs Consider consultation with psychiatrist |

slower in its evolution and usually associated with some primary infectious lesion on the extremity

Treatment—See p 3256

INFECTIOUS ECZEMATOID DERMATITIS

Infectious eczematoid dermatitis is an inflammatory eruption composed of patches of *erythema vesicles pustules* and *scaly areas* It usually results from contamination of the skin by purulent discharges and is most often observed about *chronic infectious foci* with discharge containing staphylococci Thus otitis media draining wounds and abscesses are often the source of the purulent discharge

The eruption spreads out from the infectious focus in an irregular fashion There are areas of *erythema* and *vesicles* or *pustules* are usually present The vesiculopustular lesions rupture dry and are followed by scaling New lesions continue to appear peripherally trauma and scratching producing new portals of entry for the pyogenic cocci Some degree of *itching* is usually present and also moderate regional *lymphadenitis*

The condition must be differentiated from *dermatitis venenata* (p 3330) and *pyoderma* The presence of a primary focus of infection with a purulent discharge is the important differential point

Treatment—See p 3256

DERMATITIS PAPILLARIS CAPILLITH (ACNE KELOID)

Acne keloid is an unusual disease occurring in the *nuchal region* in males and resulting from *staphylococcal infection* of the hair follicles and deeper tissues It is most common in Negroes

The disease begins on the nucha but may spread over the occiput even to the vertex It commences with many small *papulopustular* lesions which extend deeply and produce *abscesses* As healing takes place larger and smaller red hard *keloids* are formed The hair in the affected area is lost permanently

The most effective measure in the pre keloid stage is roentgen therapy

 DIFFERENTIAL DIAGNOSIS OF

Dermatoses of the Scalp and Neck

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|----------------------------------|---|
| Alopecia | Baldness (p 3440) |
| Angiomas | Particularly strawberry marks at nape of neck and port wine stains (p 3200) |
| Carbuncle | Extensive pyoderma usually situated at back of neck Of staphylococcal origin Institute intensive treatment with penicillin (p 106) |
| Contact dermatitis | Hyper sensitivity to hair-dyes and hat bands particularly Confirm with patch test (p 3330) |
| Culis verucis gyrata | Anomaly of scalp with ridging and furrowing (p 3150) |
| Dermatitis papillaris capillitis | Papulopustular lesions of neck With residual keloids after healing Especially found in the Negro (p 3255) |
| Dermoid cysts | Founded tumors encountered in median line Advise excision (p 3935) |
| Ectodermal defects | Developmental anomaly Characterized by dystrophies of hair nails skin and teeth Usually accompanied by depression of nose (p 3150) |
| Favus | Fungous infection of scalp with cup-shaped yellow dry crusts Identify fungus by spread or culture (p 3294) |
| Furuncles | Often seen along collar line as isolated pyoderms (p 3248) |
| Lichen scrofulosus | Follicular papules of trunk or neck in children A tuberculid Get biopsy (p 3269) |
| Moles | Pigmented and non pigmented hairy and non hairy nevi (p 3204) |
| Neurodermatitis | May be diffuse or localized Associated with itching and lichenification May be of allergic or psychogenic origin Look for offending allergen Consider consultation with psychiatrist |
| Pediculosis capitis | Infestation of scalp hair by head louse Note intense itching and secondary changes due to scratching and excoriation Associated posterior cervical lymphadenopathy Identify nits or lice (p 3183) |
| Perfume dermatitis | Erythema and pigmentation of neck Due to ultraviolet sensitivity conditioned by oil of bergamot in perfume (p 3177) |
| Psoriasis | Scaling papules associated with intense itching Often located in scalp Associated with more characteristic eruptions on elbows knees and extensor surfaces of the body Characterized by chronicity with exacerbations and remissions (p 3417) |
| Sebaceous cysts | Rounded masses freely movable except at point of attachment to skin Puncture yields caseous material Treat by excision (p 3208) |

- 3 The lesion is in a danger zone particularly on or above the upper lip
- 4 The lesion is deep seated as the carbuncle in any location
- 5 The lesion is recurrent as in furunculosis
- 6 The lesion is refractory and indolent as in Sycosis barbae
- 7 There is a suggestion of systemic invasion as manifest by chills fever or leukocytosis
- 8 A bacteremia is demonstrable
- 9 There is a suggestion of metastatic furunculosis particularly in lungs (p 2212) or kidneys (p 2359)

When the wisdom of initiating systemic therapy is debatable the decision should favor positive action. Our preference is a course of intramuscular injections of penicillin using an initial dose of 50 000 to 100 000 units and maintenance doses of 20 000 to 50 000 units every two or three hours. The ambulatory patient may be given an oil wax penicillin depot or sulfa therapy using an initial 2 to 4 gm of sulfadiazine followed by 0.5 to 1 gm every three hours for maintenance. As elsewhere emphasized sulfonamide therapy must be associated with a copious fluid intake and sufficient bicarbonate to alkalinize the urine (p 88).

The patient who is desperately ill or unresponsive to the simpler methods of systemic therapy requires the institution of an intravenous drip. Into this may be placed daily 500 000 to 1 000 000 units of penicillin with 5 to 8 gm of sodium sulfadiazine. Additionally 500 cc of citrated blood are added if indicated. Meantime efforts are made to combat or control associated maladies particularly diabetes mellitus and the avitaminoses. It is of particular importance to continue antibiotic therapy until a considerable period has passed following the subsidence of the graver symptoms. Premature discontinuance of therapy may lead to recrudescence with an organism sensitized to the antibiotic agency particularly the sulfonamide.

Prophylactic Systemic Treatment—Preventive treatment is required for all pyodermas. In addition to continuance of the measures outlined for active local and systemic therapy immunization with autogenous vaccine or staphylococcus toxoid may be tried (p 77). Those whose occupations result in maceration of the hands or exposure to dirt or chemicals are advised to wear gloves or change jobs. We have seen no encouraging results from the use of tin arsenic or vitamin A preparations. Continued use of sulfonamide ointment invites a treatment dermatitis. Protracted use of penicillin may lead to organism fastness (p 106).

GONOCOCCUS INFECTIONS

Gonorrheal urethritis (p 219) is rarely associated with skin lesions. In gonococcemia (p 219) *petechial lesions* are seen as in bacteremia of other types.

KERATOSIS BLENNORRHAGICA

A rare manifestation of gonorrheal urethritis and arthritis is the appearance of keratosis blennorrhagica characterized by a vesiculopustular eruption terminating in crusted scaling lesions. This complication is seen almost

Treatment of Pyodermas

The treatment of the pyoderma requires local and systemic measures aimed at abortion, localization or drainage of the presenting lesion and prevention of spread or recurrence by auto inoculation.

Isolation—Of the pyodermas only impetigo contagiosa is reportable requiring isolation. This measure is dictated by the invasiveness and virulence of epidemics in nurseries, institutions and schools.

Local Prophylaxis—Local hygiene is of paramount importance in the prophylaxis of the pyodermas. Dishwashers and others whose hands become macerated through prolonged immersion in water should wear gloves while at work. After work, skin surfaces should be scrubbed vigorously with soap and water, thoroughly dried and dusted with talcum. Reliance should not be placed on antiseptics and disinfectants.

Once the pyoderma is established, it must not be squeezed and pressed lest the organisms be disseminated in the deeper tissues. Auto inoculation is prevented by scrubbing the hands after contact with the lesion.

Abortive Treatment—Of the pyodermas the furuncle is best adapted to abortive treatment. The central hair is epilated with a forceps. Into the orifice is plunged a pointed applicator previously dipped in pure phenol after which a protective dressing is applied.

Localization—When abortive treatment fails, efforts are instituted to favor localization and hasten suppuration. The involved area is cleansed with soap and water and, if necessary, shaved. Crusts are removed particularly in impetigo contagiosa.

Hot compresses are applied every few hours for at least twenty minutes. A solution of penicillin may be improvised for this purpose by dissolving the contents of a vial containing 100,000 units in 1000 cc. of boiled water. The more recently introduced antibiotic bacitracin may be used for penicillin-resistant lesions. White Petroleum Jelly is applied to non-infected areas.

Between compresses a protective dressing is applied after application to lesions of an ointment of penicillin (1 cc. = 500 to 1000 units). The dressing is preferably fixed with gauze bandage or scotch tape if possible, avoiding adhesive plaster.

Furuncles on or above the upper lip and all carbuncles require more intensive local treatment by exposure to actinic or roentgen rays.

Surgical Drainage—Emphasis on non-operative therapy must not overshadow the vital importance of surgical incision and drainage. As soon as frank suppuration has occurred, laudable pus is released by intelligently planned incision and adequate drainage. Details of operative therapy appear in the section devoted to *Minor Surgery* (p. 3909). Needless to state, the preoperative routine is resumed immediately after drainage has been established, since premature cessation invites recurrences.

Active Systemic Treatment—Local measures must be supplemented by systemic treatment under the following circumstances:

If

- 1 The patient is a newborn infant or young child
- 2 The host resistance is lowered as in cachexia, senility or uncontrolled diabetes mellitus

Factors—Some of the lesions are caused by *exogenous* contact whereas others are *hematogenous* or *lymphogenous*. The organism involved may be of *human*, *bovine* or *avian* origin (p 252) and may vary significantly in virulence and in dosage. The reaction type is influenced by the *layer of the skin* that is attacked. The cutaneous phenomena are dependent upon whether hair follicle, lymphatic vessel, blood vessel or corium is affected. The *allergic state* determines the quality of many of the reactions to the tubercle bacillus. Patients with cutaneous *hypersensitivity* (p 262) respond quite differently from those who have never previously had contact with the organism.

THE TUBERCULOUS CHANCER (PRIMARY TUBERCULOUS COMPLEX)

The tuberculous chancre occurs in individuals who have never developed a primary pleuropulmonary or glandular infection (Ghon tubercle) and who are tuberculin negative. Since most adults have already had a primary infection the tuberculous chancre is most frequently, if not exclusively, encountered in children. The lesion may occur anywhere on exposed surfaces. An abrasion, trauma or ritual circumcision may serve as a portal of entry.

The Lesion—The tuberculous chancre is an indurated inflammatory swelling which *ulcerates*. It is accompanied by an indolent regional *lymphadenitis* and thickened lymphatic vessels. At the outset the *tuberculin test* (p 262) is negative but after a variable number of weeks it becomes positive. In many instances *spontaneous healing* occurs in several months although the lesion may take on the features of *lupus vulgaris* (p 3262).

Histopathology—The histopathology is typical of tuberculosis. There are tubercles consisting of masses of epithelioid cells surrounded by lymphocytes and an area of central necrosis. Giant cells and tubercle bacilli are present.

Diagnosis—The condition may require differentiation from the chancre of syphilis from sporotrichosis and tularemia. In *syphilis* (p 3278) the progress is more rapid, the patient is usually older and spirochetes are present in the sore and in the regional lymph nodes. In *sporotrichosis* (p 3311) the regional lymph nodes are not affected and the secondary nodules develop along the line of the lymphatic vessels. With *tularemia* (p 323) there are systemic symptoms, the lymph nodes often suppurate and *skin* (Foshay) and *serum reactions* become positive.

Local Treatment—In some instances spontaneous healing occurs so promptly that local treatment is not needed. In resistant examples *excision* of the lesion and destructive methods by *electrocoagulation* or *cauterization* have been practiced successfully.

TUBERCULOSIS VERRUCOSA CUTIS

Tuberculosis verrucosa cutis is due to a *primary inoculation* of the skin by the tubercle bacillus. The lesion is a circumscribed, elevated patch whose surface is studded with wartlike excrescences. Pustular lesions may also be present. The color is grayish and dull red and as a rule there is some redness about the periphery of the lesion.

The most common site is the *dorsum* of the hand. A variant of this lesion is the *postmortem* or *anatomic tubercle* (*verruca necrogenica*).

exclusively in males and preponderantly in the age group of twenty to forty years

The Eruption—The eruption usually appears subsequent to the development of the arthritis within the space of a few weeks or months. It commences as a *vesicle* or *papule* which soon becomes pustular, ruptures and forms a dry, adherent *crust*. The crust may be yellowish to brown in color and often tends to be conical. When removed it uncovers a raw, eroded surface. Lesions vary in number and size. There may be one to two or dozens and they may be from 1 mm. to 3 or 4 cm. in diameter.

A very common localization is the *soles* but lesions are also frequently observed on the *dorsa of the feet*, the *hands*, *extremities*, *penis*, *face* and *scalp*. Rarely they are seen in the mouth and on the conjunctiva or cornea. At times they involve the periungual and subungual regions and lead to distortion or destruction of the nail.



Fig. 930—Keratosis blennorrhagica (Courtesy of Dr. Stanley O. Chambers)*

Diagnosis—It is not possible to find gonococci in the skin lesions. However, the concurrence of the characteristic eruption with arthritis and gonorrheal urethritis suggests the diagnosis. Differentiation from *psoriasis arthropathica* (p. 3418) may be difficult. Psoriasis is more long-standing; the lesions are somewhat different morphologically and have other distribution; females are often afflicted; the mouth and eye are never affected and gonorrheal urethritis is usually absent.

Treatment—The most successful therapy is that of the underlying condition. Excellent results have been obtained with penicillin or a combination of *hyperthermia* (p. 3789), *sulfathiazole* (p. 88) and large doses of *vitamin A* (50,000 to 100,000 units daily). Nutrition should be maintained in good status and *transfusions* given if the condition warrants.

INFECTION WITH THE TUBERCLE BACILLUS

A number of cutaneous entities are produced by infection with the tubercle bacillus. The variations in these types of reaction depend on many

| Disturbance | Type of Lesion ¹ | Herald Site | Spread | Duration from Evolution to Involution | Specific Features |
|--------------------------|--|--------------------------|---------------------------------|---------------------------------------|--|
| Dengue | Macular | Dorsum of hands and feet | Extremities face and trunk | 2 to 3 days | Epidemic |
| Measles | Pale macular and maculopapular (Fig 65 p 411) | Behind ears | Face neck trunk and extremities | 3 to 10 days | Koplik spots on buccal mucosa (Fig 64 p 410) |
| Rubella | Maculopapular or erythema (Fig 66 p 417) | Face | Body and extremities | 1 to 3 days | Postcerv lymph adenopathy |
| Fourth Disease | Diffuse erythema | Face | Body and extremities | 2 to 3 days | Negative Schultz Charlton |
| Fifth Disease | Macular | Face | Body and extremities | 2 to 10 days | No Koplik spots |
| Sixth Disease | Erythema or maculopapular but not raised | Neck and trunk | Spare nose and cheeks | 1 to 2 days | No Koplik spots Negative Schultz-Charlton |
| Chickenpox | Macules papules and then vesicles (Fig 67 p 421) | Trunk | Head, face and extremities | 2 to 7 days | Pleomorphic ² Monolocular ³ |
| Smallpox | Papular vesicular and pustular (Fig 68 p 424) | Forehead and wrists | Face arms, trunk and legs | 4 to 10 days | Monomorphic ² Multilocular ³ |
| Infectious Mononucleosis | Macular papular erythematous or vesicular | Irregular | Irregular | Irregular | Lymphadenopathy Hemogram (p 469) |
| Dermatitis Medicamentosa | Macular papular vesicular urticarial (Fig 92, p 350) | Irregular | Irregular | Irregular | History of medication |

NOTES

¹ The terms morbilliform, scarlatiniform and rubelliform are avoided. The morbilliform and rubelliform rashes are maculopapular; scarlatiniform eruptions are first punctate and then erythematous.

Pleomorphic indicates that the individual lesions are simultaneously in different stages of evolution. Monomorphic means that all are simultaneously papular, vesicular or pustular.

Monolocular means the lesion can be evacuated by a single pin prick. The multilocular vesicle or pustule cannot be emptied by a single puncture.

DIFFERENTIAL DIAGNOSIS OF

Generalized Rashes in Eruptive Fevers

Before entering into a discussion of scarlet fever the first of the exanthems to be treated in this section it seems fitting to deal with the broader problem of the differential diagnosis of generalized rashes in the febrile and afebrile

The day has long since passed when the practitioner can regard the acute exanthems as consisting of measles scarlet fever rubella and chickenpox. He has since learned that a generalized eruption may be an integral part of meningococcemia infectious mononucleosis spirochetal invasions (especially syphilis) or rickettsial disease (such as typhus and spotted fever). Difficulties in diagnosis are further increased by the fact that fever and a rash may result from the administration of drugs taken by self-medication or prescription in an otherwise non-eruptive infection such as the common cold or influenza.

Conscious of the many facets to the problem the clinician therefore approaches the diagnosis of the febrile rash with caution and humility avoiding such obvious booby traps as the false positive Wassermann reaction in infectious mononucleosis and the morbilliform or scarlatiniform outbreaks caused by analgesic antipyretics sedatives sulfonamides and arsenicals. He realizes that even the expert cannot differentiate eruptions by morphology alone and he does not refrain from collecting all available data before he commits himself to a definite opinion and therapeutic program.

| Disturbance | Type of Lesion ¹ | Herald Site | Spread | Duration from Evolution to Involution | Specific Features |
|------------------------------|---|---------------------------|---------------------------------|---------------------------------------|---|
| Scarlet Fever | Punctate lesion with confluence to diffuse erythema | Neck chest and skin folds | Arms and legs Circumoral pallor | 2 to 6 days | Schultz-Charlton reaction (Fig 18 p 164) |
| Meningococcemia | Maculopapular petechial or purpuric (Fig 23 p 212) | Body | Shoulders and thighs | 2 to 5 days | Headache and meningeal symptoms |
| Tularemia | Maculopapular and pustular | Body | Irregular | Irregular | Handling rodents Tick bite |
| Syphilis | Maculopapular erythema vesicular or pustular (Fig 49 p 338) | Irregular | Irregular | Irregular | History of chancre (p 335) Positive dark field and serology (p 337) |
| Pat Bite Fever | Maculopapular | Chest and arms | Irregular | Irregular | History |
| Typhus Fever | Macular and petechial (Fig 57 p 370) | Axilla and loins | Abdomen chest and back | 2 to 10 days | Epidemic Weil-Felix reaction (p 372) |
| Rocky Mountain Spotted Fever | Maculopapular and petechial (Fig 58 p 378) | Wrists ankles | Scalp chest abdomen | 2 to 3 weeks | Tick bite |

The disease is extraordinarily chronic with slight tendency to spontaneous cure. It usually starts in childhood and the lesion may still show activity and spread four and five decades later.



Fig. 951. A Scrofuloderma with lupus vulgaris. B Lupus vulgaris.



Fig. 952.—Lupus miliaris disseminatus faciei.



Fig. 953.—Erythema induratum (Bazin).

Fig. 954.—Sarcoidosis.

The Lesion—At the outset lupus appears as a single reddish brown tiny *papule*. Other similar papules appear in the immediate vicinity and gradually coalesce until the characteristic picture emerges as a flat slightly elevated *patch* that is shiny reddish brown and scaling. Centrally as well as

which is similar in appearance but smaller in size. It is most often seen on the hands of those working with human cadavers and in butchers.

Pathology—The histopathology is similar to that seen in *lupus vulgaris* (p. 3965). In the cutis there are many tubercles consisting of epithelioid cell masses surrounded by a mantle of lymphocytes and giant cells. Tubercle bacilli may be present but caseation necrosis is absent.

Diagnosis—The indolence, location and appearance of the lesion make it striking and easy to recognize. Differentiation from *blastomycosis* (p. 493) may be difficult on clinical grounds without bacteriologic and histopathologic study. The lungs are investigated to determine the presence of active disease.

Treatment—Treatment is chiefly local. Small lesions are excised. Those not amenable to surgery are destroyed by *cautery*, *electrocoagulation* or applications of *pyrogallic acid* (p. 3125). *Roentgen therapy* is useful in some cases.

SCROFULODERMA (TUBERCULOSIS CUTIS COLLIQUATIVA)

Scrofuloderma is a tuberculous infection of the skin in which the tubercle bacilli have been implanted from a primary focus subjacent to the skin, such as tuberculous lymph nodes or infections in the bones or joints. This form of tuberculosis is seen chiefly in childhood when lymph node and bone tuberculosis are most frequent.

The Lesion—The condition begins with localized *subcutaneous swellings* which become adherent to the skin. The skin becomes dusky red and infiltrated; softening appears at one or more points and *ulceration* occurs. There is discharge of purulent matter but the ulcer remains its walls infiltrated and extending somewhat peripherally. (Fig. 952 A.)

Diagnosis and Pathology—The diagnosis is simple, based upon the history and the location of the process over areas of involved lymph nodes, diseased bone or joint. The *histopathology* is very similar to that seen in tuberculous chancre but the *tuberculin test* is usually strongly positive.

Treatment—Local treatment consists of *ultraviolet irradiation*, *roentgen therapy*, local applications of *cod liver oil* (p. 3117) and graded intradermal injections of *tuberculin* (p. 262).

TUBERCULOUS ULCERS (TUBERCULOSIS CUTIS ORIFICIALIS)

Tuberculous ulcers are painful lesions which affect any portion of the mouth and also may appear upon the skin adjacent to the natural orifices (mouth, urethra, vagina, anus). They are superficial, unhealthy sores characterized by exquisite tenderness and painfulness and are due to direct implantation of the bacilli from an adjacent focus in a viscus.

The *histopathology* is similar to that of tuberculous chancre with numerous bacilli. There is marked hypersensitivity to tuberculin.

Treatment of the local lesion is of secondary importance, the visceral lesion being the vital concern. The ulcers have been treated successfully by *electrodesiccation*.

LUPUS VULGARIS (TUBERCULOSIS CUTIS LUPOSA)

Lupus vulgaris is the most common form of cutaneous tuberculosis. The sites of predilection are the *cheek* and *nose*, though patches are seen on the trunk and extremities alone or in conjunction with the facial lesion.

Syphilis (Neonatal)

Seropurulent or sanguineous nasal discharge (snuffles). Usually associated with mucous patches in mouth and other stigmas of infection. Get darkfield examination and Wassermann (p. 2787).

Syphilis (Tertiary)

Saddle nose or perforation of septum from breaking down of gumma. Occasional gummatous lesion consisting of rounded painless tumor of tip of nose or nasal septum. Get Wassermann and note therapeutic response to iodide (p. 608).

peripherally appear distinct brownish *papules* or *nodules* pressure upon which with a glass slide reveals an infiltrate whose color resembles that of apple jelly. The lesion of lupus vulgaris may *ulcerate* undergo considerable *hypertrophy* or form *papillary excrescences* (Fig. 952 B).

When the nose is attacked there may be marked destruction of the soft parts with ugly disfiguration although the bony structure is spared.

Histopathology—The histopathology is characteristic with noncaseating epithelioid tubercles in the cutis. Tubercle bacilli are present in small numbers. The tuberculin skin test is positive. Active pulmonary tuberculous disease is rarely present but the primary infection (Ghon tubercle) is demonstrable.

Diagnosis—The lesion of lupus vulgaris may need to be differentiated from late cutaneous *syphilis* (p. 3286) of the nodulo ulcerative form. The most important morphologic difference is the presence of active nodules in the central portion of the lesion whereas in syphilis the center becomes scarred and inactive. The serologic tests further clarify the situation.

In *lupus erythematosus* there are no apple jelly nodules the lesion is of shorter duration usually starting in adult life and the histopathology is completely different.

Treatment—Recommended for local therapy are exposure to *Finsen* or *Kromayer* light and destruction by *electrodesiccation* *coagulation* or 5 to 10 per cent *Pyrogallic Acid* (p. 3125). Small lesions may be excised exposed to *roentgen treatment* or destroyed with *carbon dioxide snow* (p. 3785).

In addition to local treatment certain systemic measures are recommended. The administration of a high calory high vitamin diet cod liver oil and generalized tonic dosages of ultraviolet light are useful. Success has been claimed from intravenous injections of *Gold Salts* (p. 2922) and graded intradermal injections of *tuberculin* (p. 262). Daily doses of 50,000 to 150,000 I.U. of calciferol are reported to yield excellent results.

TUBERCULIDS

In the diseases described in the foregoing paragraphs tubercle bacilli are demonstrable in tissue sections culture media or by animal inoculation. The tuberculids form another group of morphologic entities whose tuberculous etiology has been adequately confirmed despite the fact that bacilli are rarely demonstrable. These tuberculids include lichen scrofulosorum papulonecrotic tuberculids lupus miliaris disseminatus faciei rosea cea like tuberculid of Iewandowsky erythema induratum and sarcoidosis.

DIFFERENTIAL DIAGNOSIS OF

Dermatoses of the Nose

Dermatoses of the nose constitute cosmetic blemishes that cause a great concern to sensitive patient. Pyoderms present the threat of invasion of venous channels with subsequent bacteremia and intracranial complications.

CLINICAL MANIFESTATIONS AND
DIAGNOSTIC FEATURES

| | |
|---------------------|--|
| Acne Rosacea | Persistent redness with acneform lesions and telangiectases. Occurs in adults (p 3357) |
| Acne Varioliformis | Papulopustular lesion in patients beyond the age of 30. No comedones (p 3356) |
| Acne Vulgaris | Papulopustular affliction of adolescents with oiliness of skin and comedones. Tendency to recurrence (p 3358) |
| Adenoma Sebaceum | Congenital papular lesions of nose often associated with telangiectases (p 3148) |
| Chilblain | Tense elevated and erythematous lesions with severe burning and itching. Follows exposure to cold (p 3173) |
| Comedones | Blackheads. Frequently seen in adolescent with acne vulgaris |
| Epithelioma | Isolated elevated waxlike papule with tendency to ulceration. Usually occurs in males beyond the age of 40. Get biopsy (p 3220) |
| Erysipelas | Acute streptococcal infection associated with intense pain and systemic manifestations. A fiery red lesion with raised advancing border (p 167) |
| Erythema Simplex | Redness of nose. Usually congenital but may be associated with chronic alcoholism |
| Furunculosis | Intensely painful acute pyoderm (p 3248). Usually involves skin of ala or hair follicles within nostrils |
| Impetigo Contagiosa | Pyoderm with crusting lesion (p 3251). Most often encountered in infants and children |
| Lupus Erythematosus | Persistent irregular patches of redness with dry adherent scales. Removal of scales reveals keratotic prolongations into dilated sweat ducts. Telangiectases often present. Lesion may be solitary or form characteristic butterfly pattern. After healing depressed and atrophic macule remains. May be associated with systemic manifestations including endocarditis, diffuse vascular lesions and nephropathy. Get biopsy (p 3399) |
| Lupus Vulgaris | Cutaneous form of tuberculosis with brownish apple-jelly papules or nodules. May ulcerate with marked destruction and scar formation. Get biopsy (p 3262) |
| Rhinophyma | Chronic bulbous hypertrophy of tip of nose. Occurs most frequently in middle-aged men with acne rosacea and seborrheal dermatitis (p 3357) |
| Rhinoscleroma | Chronic low-grade granulomatous disease involving external nose and nasal cavity |

| | |
|------------------------------|--|
| Dermatitis medicamentosa | Drug eruptions of various types due to hypersensitivity to therapeutic agents. Check history (p 3335) |
| Dermatosis papulosa nigra | Congenital dermatosis in which pigmented papules appear on the middle third of the face in Negro women (p 3150) |
| Dermatomycoses | Fungous infections such as actinomycosis. With chronic granulomas and ulceration. Identify pathogen by smear and culture (p 3293). Start antibiotic therapy. |
| Disseminated neurodermatitis | Discolored plaques associated with excoriations from marked itching. Usually of skin folds or other flexural areas. Probably an atopic dermatitis. Seek offending allergen (p 3343) |
| Epitheliomas | Of basal or squamous cell varieties. Former usually above upper lip. Latter may be of lower lip with the production of chronic ulcers with rolled borders. A cutaneous malignancy. Get biopsy (p 3220) |
| Erysipelas | Streptococcal infection with advancing fiery red border, intense pain and constitutional manifestations. Isolate pathogenic streptococcus. Initiate systemic treatment with penicillin (p 106) |
| Exanthems | Systemic eruptive fevers with erythematous or scarlatiniform rashes (p 180), maculopapular eruptions (p 412) or vesicular and pustular lesions (p 422) |
| Foot and mouth disease | Virus infection with vesicles and pustules appearing in the neighborhood of mouth and on feet. Associated with lymphadenopathy. Appears in epidemic form (p 437) |
| Fordyce's condition | Depositions of tan pigment of cheeks and lips (p 3465) |
| Freckles | Normal pigmentary responses to exposure to sun. |
| Furuncles | Isolated or multiple staphylococcal infections. |
| Granulosis rubra nasi | Redness and shininess of the nose. Congenital disturbance noted in childhood. Often associated with localized sweating (p 3464) |
| Herpes simplex | May be associated with exposure to sunlight. Acute upper respiratory infections, lobar pneumonia and meningococcemia (p 433) |
| Herpes zoster | Painful vesicular dermatosis. With ominous ophthalmic complications when 5th cranial nerve is involved (p 435) |
| Hydroaestivale | Congenital dermatosis of childhood with the appearance of facial vesicles during the summer (p 3176) |
| Impetigo contagiosa | Pustular dermatosis of childhood and adult life with crusting and spread by continuity. Note response to local treatment with sulfathiazole or penicillin (p 3251) |
| Keratosis follicularis | Congenital dermatosis of infancy and childhood with the appearance of papules with greasy crusts. Often of the face. Try therapeutic response to vitamin A (p 3153) |

DIFFERENTIAL DIAGNOSIS OF

Dermatoses of the Face

See also Dermatoses of the Beard p 3437 Dermatoses of the Ears p 2113 Dermatoses of the Nose p 2110

CAUSES

Acne rosacea

Acne varioliformis

Acne vulgaris

Achnomycosis

Adenoma sebaceum

Angiomas

Angioneurotic edema

Anthrax

Atopic dermatitis

Avitaminosis

Carbuncle

Chapping

Chilblain

Chloasma

Comedones

Contact dermatitis

Cutaneous horn

CLINICAL MANIFESTATIONS AND
DIAGNOSTIC FEATURES

Erythema and telangiectases of middle third of face in adults (p 3357)

Pustules of face with necrosis and scarring in adults (p 3356)

Comedones and pustules of face neck and shoulders in adolescents (p 3358)

Lumpy jaw Chronic granuloma with multiple sinuses Identify ray fungus in smears and cultures (p 489) Start intensive antibiotic therapy

Yellow and deep red papules with telangiectases of middle third of face Congenital dermatosis of childhood Associated with tuberous sclerosis and organic neurologic disturbances (p 3148)

Strawberry and port wine marks Congenital anomalies (p 3200)

Sudden localized swellings with burning and itching Note therapeutic response to epinephrine Seek offending allergen (p 3349)

Malignant pustule often the result of contamination from new shaving brush Identify gram positive spore bearing bacillus by smear and culture (p 292) Start intensive therapy with penicillin.

Infantile eczema An allergic dermatosis often due to hypersensitivity to digestants such as milk and eggs (p 3342)

Rhagades of margins of lips in riboflavinosis Thickening and pigmentation of face Often of butterfly distribution in pellagra Note therapeutic response to vitamin B complex

Grave staphylococcal infection often involving lips nose cheeks or eyelids (p 3249) Start intensive treatment with penicillin

Particularly of lips due to cold

Redness burning and later vesiculation due to exposure to low temperatures Consider use of anticoagulants

Pigmented mask of pregnancy (p 2625)

Blackheads Usually associated with acne vulgaris

Often the result of exposure to poison ivy soap cosmetics lip stick, perfumes and nail polish Check history Verify with patch test. Yellow to black tumors associated with advancing years Get biopsy May be a precancerous (p 3217)

| | |
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| Rosacea like tuberculid of Lewandowski | Diffuse erythema of face with brownish papules. A tuberculid appearing in women. Get biopsy (p 3270) |
| Sarcoidosis | Systemic disorder probably tuberculous with the appearance of brownish nodules or plaques especially on face. May be associated involvement of uvea and salivary glands. Get biopsy (p 3263) |
| Scleroderma | Systemic disturbance with the appearance of a hide bound skin (p 3428) |
| Sebaceous cyst | Isolated non inflammatory tumors attached at one point to the skin. Evacuation yields caseous material (p 3208) |
| Seborrheal dermatitis | Greasy scales and pigmentary disturbances associated with seborrhea of the scalp. Often coronal in distribution. May involve middle third of face (p 3432) |
| Senile sebaceous adenoma | One or several yellow umbilicated papular lesions usually of forehead. May be a precancerous. Get biopsy (p 3205) |
| Solar dermatitis | Sunburn |
| Syphilis | Primary secondary and tertiary lesions may appear. Supplement serologic test with dark field microscopy (p 45) |
| Tinea | Superficial ring worm with tan scaling macules. Identify fungus by smear and culture (p 3293) |
| Tularemia | Primary lesions may appear on face. In association with lymphadenopathy. Isolate pathogen by smear and culture (p 323) |
| Urticaria pigmentosa | Rare dermatosis with whealing of pigmented macules when rubbed. Look for specific allergen (p 3158) |
| Verrucae | Warts. Excise if possible |
| Xanthoma | Yellow papules or nodules associated with diabetes mellitus or the systemic reticuloendothelioses. Check blood sugar and blood cholesterol. Make complete physical examination (p 3244) |
| Xeroderma pigmentosum | Photosensitivity of face and other exposed parts resulting in pigmented macules, areas of atrophy, telangiectases and keratoses. A precancerous. Get biopsy (p 3158) |

The relationship of the tubercle bacillus to sarcoidosis is still a moot question.

Lichen Scrofulosorum (*Tuberculosis Cutis Lichenoides*)—Lichen scrofulosorum is an eruption seen in children consisting of grouped rounded follicular *papules* skin colored or slightly darker occurring most often on the trunk or neck. There may be one or several such groups of lesions. They appear more or less abruptly and disappear spontaneously. A search for a primary focus in the lungs or elsewhere should be made. The *tuberculin skin test* is usually strongly positive.

Differentiation from *keratosis pilaris* and lichen planus may be necessary. The distribution of *keratosis pilaris* (p 3236) is usually on the arms

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| Keratosis | Pigmented scaly warts occurring in association with seborrhea and advancing age. A precancerous. Get biopsy (p 3166) |
| Leishmaniasis | A tropical ulcerating dermatosis often involving the face. Identify Leishman Donovan bodies in direct smears (p 48) |
| Leprosy | Anesthetic macules and nodules of face. Get biopsy (p 273) |
| Lupus erythematosus | Scaling papular eruption of the middle third of the face. Most frequently seen in females. Note increase at margin and atrophy in center. May assume butterfly distribution over bridge of nose. Often associated with telangiectases and constitutional symptoms involving particularly the heart valves, peripheral vessels and kidneys (p 3397) |
| Lupus miliaris disseminatus faciei | A tubercloid in which brownish papules appear on the face. Get biopsy (p 3263) |
| Lupus vulgaris | Apple jelly nodules of cheek or nose with ulceration and destruction of tissue. A tubercloid. Get biopsy (p 3263) |
| Milium | Rounded white nodules appearing on the face of young people (p 3403) |
| Moles | Pigmented and non pigmented hairy and non hairy nevi (p 3204). Excise if possible |
| Molluscum contagiosum | A virus skin disease characterized by the appearance of umbilicated vesicles containing cheesy material. Most often seen on face, trunk and male genitals (p 3257) |
| Multiple benign cystic epitheliomas | Rare familial disease of females with lentil sized cysts appearing over face. Get biopsy (p 3207) |
| Multiple hemorrhagic familial telangiectases | Hereditary and familial disease with multiple vascular anomalies of skin and internal organs. Get blood count and group for transfusion if necessary (p 3394) |
| Paraffinomas | Irregular tumors due to the injection of paraffin in attempted cosmetic surgery |
| Pediculosis | Infestation of scalp and eyelashes by lice. Look for nits on hairs as well as lice or crabs |
| Pemphigus | Fetal dermatosis with itching of the skin followed by the development of bullae involving the entire surface as well as the mucous membranes. Get biopsy (p 3405) |
| Perleche | Chronic dermatosis of lip with fissuring. Often due to association of vitamin B deficiency and monilia invasion. Isolate pathogen. Note therapeutic response to vitamin B complex (p 1696) |
| Pustular folliculitis | Of bearded area with areas of suppuration surrounding hair follicles (p 3247). Note local response to sulfathiazole |
| Rat bite fever | Primary lesion often on face. Get history. Attempt isolation of pathogen (p 362). Treat with penicillin |
| Rhinophyma | Massive enlargement of nose with pitting. As manifestation of acne rosacea (p 3357) |

Erythema Induratum (*Tuberculosis Indurativa Subcutanea* Bazin's Disease) — *Erythema induratum* is met with in *females* from the ages of thirteen to thirty five. It consists of purplish red dense inflammatory *nodules* on the *calves*. There is usually little pain or disability. The nodules invariably break down and eventuate in indolent punched out *ulcers* (Fig. 953).

Active pulmonary or other forms of tuberculosis are generally absent but the primary infection (p. 259) in the lung is invariably found and tuberculin hypersensitivity is well marked.

Diagnosis — The syphilitic gumma (p. 3286) is rarely bilateral is more common in the upper third than the lower third of the leg and is seen in older persons. Serologic tests for syphilis are positive and therapeutic response differs. *Erythema nodosum* (p. 3377) occurs on the front of the legs the lesions are tender and painful and never ulcerate. *Ulcers associated with varicose veins* are very uncommon in young persons and are usually unilateral the leg is edematous and the lesions are rarely situated on the calves.

Treatment — Most important in therapy is *bed rest* with elevation of the leg *cod liver oil* (or ointment) locally and orally and local irradiation with *ultraviolet light*. When the patient is ambulatory supportive bandages are useful. Intradermal injections of graded doses of *tuberculin* at times are of value.

Sarcoidosis (Besnier Boeck Schaumann's Disease) — Systemic sarcoidosis (Besnier Boeck Schaumann's disease) is an uncommon constitutional disturbance believed to be produced by the tubercle bacillus. Any body tissue may be affected but the sites of predilection are the skin lymph nodes lung liver spleen bones and eyes.

The Eruption — The skin may show only a solitary lesion or many dozens may be present. In some instances there is no eruption but the disease is otherwise typical. The commonest types of eruption are *nodules* or *plaques* of varying size (1 mm. to 8 cm.). These are firm nontender and brownish and are often traversed by *telangiectatic vessels*. The face especially about the eyes nose and mouth is a favorite localization but the upper extremities neck trunk and buttocks are frequent sites. The lesions may in fact appear anywhere on the skin surface. They do not ulcerate but are gradually absorbed leaving some pigmentation and at times mild atrophy as a residuum (Fig. 954).

Clinical Manifestations — Common findings in systemic sarcoidosis aside from the skin lesions are enlargements of the superficial and deep *lymph nodes* in the *mediastinal* and *hilar regions*. Thus a picture simulating *Hodgkin's disease* (p. 1138) may be produced. Discrete deposits occur in the lower lobes of the lungs which resemble miliary tuberculosis or metastatic carcinomatosis. There is moderate enlargement of the *spleen* and *liver* *iridocyclitis* or *uveitis* and *spindle like enlargement of the phalanges*. The last mentioned is a common and distinctive alteration and has been described under the name *osteitis tuberculosa cystica multiplex*.

A special syndrome is *uveoparotid fever* in which there is involvement of the uveal tract and the parotid glands.

Laboratory Findings — There are no really significant laboratory findings. The blood count may be normal or show mild secondary anemia and leukopenia eosinophilia and monocytosis. The plasma globulin is often increased. A very characteristic and frequent condition is the presence of

and thighs and there may be associated night blindness. *Lichen planus* (p 3389) is uncommon in children it is usually pruritic, occurs on the legs and fore arms and the lesions are violaceous flat shiny polygonal papules.

Local treatment of the eruption is not required

Papulonecrotic Tuberculid (*Tuberculosis Cutis Papulonecrotica*)—The papulonecrotic tuberculid occurs in children and adults. By predilection it affects the *extensor surfaces* of the upper and lower *extremities* and at times the *face* and trunk. The lesions are discrete firm pointed *papules* whose summit soon becomes necrotic and crusted. After healing they leave a pitted *varioliiform scar*. The lesions tend to appear in crops.

Pathology—The pathology is tuberculoid with considerable nonspecific inflammation and polymorphonuclear cellular infiltration. Tubercle bacilli are not found. There is cutaneous hypersensitivity to tuberculin as a rule but this may be absent.

Diagnosis—The condition must not be confused with papular urticaria drug eruptions, acne vulgaris and acne varioliiformis. *Papular urticaria* (p 3343) is seen in children the area of central necrosis is absent itching is marked and lesions are usually much more numerous. *Drug eruptions* (p 3335), especially iodides and bromides may produce a similar lesion but distribution is generally more widespread and there is a history of ingestion of the drug. In *acne vulgaris* (p 3358) the lesions are not so disseminate there are comedones and pustular lesions but necrotic lesions are not present. *Acne varioliiformis* (p 3356) is seen in adults chiefly along the hair line and on the nose.

Treatment—The patient should be thoroughly examined for active pulmonary disease. Treatment with generalized *ultraviolet irradiation* is usually adequate.

Lupus Miliaris Disseminatus Faciei—Lupus miliaris disseminatus faciei may be considered a clinical variant of lupus vulgaris which it resembles more closely than the other tuberculids. It is seen on the face and appears as discrete brownish *papules* closely resembling the apple jelly nodules of lupus vulgaris (p 3262). Tubercle bacilli are more readily demonstrated than in other tuberculids.

The disease is seen in adults between twenty and forty years of age. Its histopathology is similar to that of lupus vulgaris but there is usually more caseation necrosis. The tuberculin skin test is generally negative but may be positive.

Treatment similar to that of papulonecrotic tuberculid is indicated (p 3270).

Rosacea like Tuberculid of Lewandowsky—The rosaceous tuberculid is a rare condition which consists of a diffuse *erythema* of the skin of the face. Studding this area are numerous pinhead sized brownish *papules*. The eruption is seen almost exclusively in *women* and must be differentiated from *acne rosacea* (p 3357) which involves the middle third of the face. The rosacea like tuberculid usually leaves this area free and involves the cheeks back to the ears.

The histologic structure is tuberculoid in character and cutaneous hypersensitivity to tuberculin is marked.

The best therapeutic results have been attained with systemic treatment. The injection of the *gold salts* (p 2922) and of graded doses of *tuberculin* intradermally have been successful.

The lesion may spontaneously shed its gangrenous slough and heal without significant constitutional symptoms. In unfavorable instances however there is invasion of the blood stream and death from metastatic infection.

The malignant pustule resembles a *carbuncle* (p 3249). However the intensity of constitutional symptoms, the presence of the gangrenous eschar and the occupation of the patient often suggest the diagnosis. Smears and cultures from the lesion reveal the presence of a Gram positive spore bearing bacillus (*B anthrax*) (p 292).

The local *treatment* consists of simple protective measures. Incision, excision and traumatization of the skin lesion are contraindicated since they may cause a hematogenous spread. The goal of the therapist is the prevention and treatment of systemic invasion by the use of *specific anti serum* (p 293) and *penicillin* (p 106).



Fig 9 5—Malignant pustule of anthrax

GLANDERS (FARCY)

The specific cutaneous lesion of glanders is the *farcy bud*. This is a skin *abscess* accompanied by an intense local reaction, particularly along the lines of the lymphatics where nodular enlargements appear and rapidly ulcerate. A thick *chocolate colored substance* is produced, the appearance of which is highly characteristic and suspicious, particularly if the disturbance has occurred in those who handle horses, mules and asses.

The *diagnosis* of farcy requires laboratory assistance through direct smears and cultures of the pus. A local skin reaction (*mallein*) is also available.

When systemic infections have occurred, a *generalized pustular eruption* often occurs and the organism (*B mallei*) can also be demonstrated in the secondary lesions.

ERYSIPELOID

Erysipeloid is a reddened, slightly elevated flat area of firm inflammation with a sharply margined border. The lesion occurs in persons who

* Courtesy of New York City Department of Health

tuberculin anergy as evidenced by complete absence of cutaneous reaction to intradermal tuberculin

Pathogenesis—The disease is most often observed from adolescence to the menopause but may occur at any age. In the United States a disproportionately large number of cases has been reported in Negroes but no race is exempt. Tubercle bacilli have on one or two occasions been isolated from fresh sarcoidal skin lesions but the relationship to tuberculosis is still a moot point. Of greatest significance is the fact that *the tuberculin test is negative* in a racial and age group where it should be positive and the fact that active pulmonary tuberculosis not infrequently supervenes. When it does the skin lesions generally disappear quickly and the skin once more reveals tuberculin hypersensitivity.

Course and Prognosis—The course is marked by periods of relapse and remission which may extend for months or years. The general health may be well maintained throughout the long years that the disease persists. As a rule the prognosis is good unless there is involvement of vital structures or pulmonary tuberculosis develops.

Diagnosis—When the entire picture is apparent the diagnosis is simple but on the basis of one or two skin lesions confusion may arise. However the *histopathology* is characteristic and biopsy should be done when necessary. The tissue reveals discrete well defined accumulations of epithelioid cells at times with giant cells but lacking the circumambient mantle of lymphocytes and the central necrosis of the true tuberculous infiltrate. These have been called naked or hard tubercles. Differentiation from *sarcoidal leprosy* (p. 276) may present great difficulty, unless there are concomitant findings such as the lepra bacillus in the nasal discharges and neural involvement.

Treatment—Treatment is empiric. Preservation of the general health, the administration of *inorganic arsenic* (Fowler's solution), a substantial diet enriched with *vitamins* and *ultraviolet light irradiation* have proved of value in many instances. Success has been obtained by the intradermal injection of *old tuberculin* starting with 0.1 cc. of a 1:100 dilution given at weekly intervals. When local reactions begin a 1:1000 dilution and later 1:10,000 solution is used, avoiding sharp local focal or systemic reactions. Improvement in the skin lesions may occur with the increase in tuberculin sensitivity. *Röntgen treatment* of the skin lesions may hasten resolution.

There is not infrequent coexistence of *syphilis* and *sarcoidosis* evidenced only by the positive serologic tests of the blood but antisiphilic treatment does not appear to benefit the sarcoidosis materially.

ANTHRAX

The malignant pustule of anthrax characterizes the portal of entry of this virulent systemic infection whose constitutional manifestation and systemic treatment are elsewhere considered (p. 292). The malignant pustule appears within three days after inoculation as a red *papule* which rapidly becomes *vesicular* and finally *purulent*. The pustule breaks and discharges leaving a black gangrenous depressed papule. The surrounding area of redness and induration increases. *New vesicles* appear about the central lesion which is usually single and is seen most commonly on the *face* and/or *forearms*. *Regional lymphadenopathy* is usually present.

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| Lymphopathia Venereum | Primary vesicle darkfield negative Later bilateral lymphadenopathy with positive Frei test (p 471) |
| Melanocarcinoma | Black malignant nodule Biopsy (p 3225) |
| Molluscum Contagiosum | Umbilicated vesicles of virus origin (p 3287) Darkfield negative |
| Moniliasis | Yeast infection in diabetics and pregnant Identify monilia (p 3301) |
| Pediculosis Pubis | Intense pruritus with identification of crabs or nits (p 3185) |
| Scabies | Intense nocturnal pruritus with characteristic burrows and excoriations Identify parasite (p 3180) |
| Senile Vulvovaginitis and Kraurosis Vulvae | Atrophy fissuring pruritus and leukoplakia in elderly females Administer estrogen (p 2597) |
| Syphilis | Secondary syphilids may be flat soft condylomas (p 338) Darkfield and Wassermann positive See also Chancre |
| Tuberculous Chancre | Chronic ulcer following ritual circumcision Get smears and biopsy (p 51) |

handle fish and meat and the causative organism is the bacillus of *swine erysipelas* (p 328)



Fig 956—A Chancroid lesion of penis B Chancroid skin test (48-hour reading)

The local process spreads peripherally and is usually unaccompanied by constitutional symptoms unlike the situation in true erysipelas (p 167)

The erysipeloid lesion tends to spontaneous recovery in two or three weeks Local treatment consists of the application of hot wet dressings and benefit has been reported from the systemic administration of sulphonamides A concentrated anti erysipeloid serum is available in 10 cc vials The recommended initial dose is 2 to 5 cc with repetition using larger

DIFFERENTIAL DIAGNOSIS OF

Dermatoses of the Genitals and Perineum

There are few problems more difficult than this. The wary practitioner must assume that the disturbance is of venereal origin unless he has definite proof to the contrary. He cannot rest content until he has done a darkfield examination of open lesions for *Treponema pallidum*, a serologic test for syphilis, the Frei skin test for lymphopathia venereum, and examined stained smears for *H. ducreyi* (chancroid) and the Donovan bodies of granuloma inguinale.

Despite these elaborate precautions experience reveals that non-venereal dermatoses of the genital area are not uncommon. Many patients suffer from lichen planus, herpes simplex, scabies, pediculosis pubis, intertrigo, dermatophytosis, non-specific vulvovaginitis, molluscum contagiosum, acuminate warts, most of which conditions present clearly recognized morphologic features.

CAUSE

DIAGNOSTIC FEATURES

Angioneurotic Edema

Urticarial lesion involving penis, vulva or scrotum (p. 3349)

Chancre

Vesicular, papular and ulcerated with induration (hard chancre) and lymphadenopathy. Darkfield positive but Wassermann negative.

Chancroid

A venereal infection due to *H. ducreyi*. Lesion macular, papular and ulcerative (soft chancre). Darkfield and serology negative. Identify organism in stained smears (p. 289).

Condylomata Acuminata

Pointed non-venereal moist papules in the uncircumcised (p. 2595). Darkfield and Wassermann negative.

Contact Dermatitis

Allergic dermatosis from condoms, venereal prophylactics, etc.

Cutaneous Horn

Precancerous warty projection. Biopsy (p. 3217).

Dermatophytosis

Fungal infection of thighs, groins and moist areas. Identify tinea (p. 3295).

Epithelioma

Usually squamous cell with ulceration and adenopathy. Biopsy (p. 3223).

Erosive Balanitis

Fusospirochetosis (p. 355) with dirty ulcerating slough. Identify Vincent organisms and refer to expert for darkfield microscopy. Wassermann negative.

Erythroplasia

Rare precancerosis with dark red plaques. Biopsy (p. 3381).

Filariasis

Tropical disease with edema of scrotum or vulva and lymphadenopathy. History of prevalence in locality (p. 3321).

Granuloma Inguinale

Progressive ulcerating venereal disease. Darkfield, Frei test and serology negative. Identify Donovan bodies by smear (p. 475).

Herpes Simplex

Simple vesicle (p. 433). Darkfield negative.

Intertrigo

Particularly in skin folds in the obese and uncircumcised (p. 3161).

Leukoplakia

White elevated precancerous plaque usually due to chronic irritation. Biopsy (p. 3213).

Lichen Planus

Multiple flat violaceous papules. Severe pruritus (p. 3383).

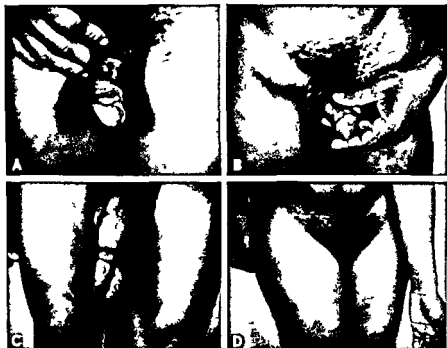


Fig 93 —Lymphopathia venereum. *A* Primary lesion of penis *B* Lesion of penis with bubo *C* Lesion of penis with elephantiasis *D* Bubos in female with positive Frei test of forearm.



Fig 938 —Granuloma inguinale Lesions of penis, groins and anus

quantities if necessary. Preliminary reports suggest also that intramuscular injections of penicillin are curative. Because of possible reactions to the specific serum, an initial trial with penicillin seems wiser (p 106)

SYPHILIS

The main discussion of syphilis appears in the section on *Infectious Diseases* (p 331). This arrangement emphasizes the concept that the af

DIFFERENTIAL DIAGNOSIS OF

Disturbances of the Penis

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|-------------------------|--|
| Physiological | Impotence. Priapism or chordee (p 2411) |
| Congenital Anomalies | Hypoplasia epipadias hypospadias or duplication (p 2280). Hermaphroditism (p 253). Cysts of raphe (p 2423). Phimosis (p 242). |
| Venereal Infections | Primary and secondary syphilis with positive darkfield and Wassermann (p 337). Chancroid with <i>H. ducreyi</i> on smears (p 2455). Lymphopatia venereum with positive Frei test (p 2457). <i>Granuloma inguinale</i> with Donovan bodies (p 2457). Gonorrhea with gram-negative cocci in urethral discharge (p 2338). |
| Non venereal Infections | Paraphimosis, balanoposthitis and non-specific urethritis (p 2454). Diphtheria, tuberculosis, fusio-purilosis and actinomycosis. Check smears and cultures (p 50). |
| Analogues of Dermatoses | Lichen planus with pruritic violaceous lesions (p 2458). Psoriasis with scale elsewhere (p 2458). Contact dermatitis usually from condom. Scabies with burrows (p 2458). Herpes with vesicle (p 435). |
| Neoplasms | Benign and malignant. Biopsy (p 2439). |
| Trauma | Contusion, rupture, strangulation, subluxation and wounds. Relieve urinary retention. Give penicillin and anti-gas gangrene serum with penetrations (p 2427). |
| Vascular Lesions | Thrombosis of dorsal vein (p 2438). Acute and chronic cavernositis (p 2454). May require aspiration or evacuation to prevent gangrene. |

fection is a *systemic disorder* rather than a localized abnormality of the cutaneous structures. The present material deals with the skin manifestations of syphilis since these are most often the presenting complaints or the physical findings that attract the attention of the patient or his practitioner.

Primary, secondary and tertiary lesions are observed in acquired syphilis, whereas only secondary and tertiary manifestations are noted in prenatal infections.

lymphadenopathy will be enabled to make this important diagnosis in the seronegative stage of the disease when curability approaches the absolute (p 340). To await the appearance of positive serologic tests means the sacrifice of the most valuable days and weeks of therapy.

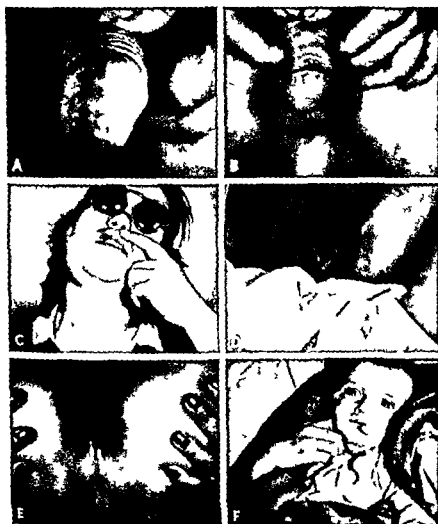


Fig. 959.—Clinical manifestations of syphilis. The primary lesion (chancre). A and B Penis. C Lip. D Vulva. E Anus. F Face in child infected by parent.

The chancre may resemble lesion of various types such as *herpes genitalis* (p 3289) *chancroid* (p 288) *erosive balanitis* (p 2456) *scabetic eruption of the penis* (p 3180) *lymphogranuloma venereum* (p 471) *granuloma inguinale* (p 475) the portal of entry of *tularemia* (p 323) a Vincent infection of the throat or tonsil or *diphtheria* (p 302) *cancer of the tongue* (p 1718) the *tuberculous chancre* (p 3259) a chronic suppurative *paronychia* (p 3973) or a *cervical neoplasm* (p 2551).

PRIMARY SYPHILIS (THE CHANCER)

The primary lesion of syphilis is the *hard chancre* which unfortunately is infrequently observed or correctly evaluated by the patient. Many patients with syphilis strongly maintain with apparent veracity that no sore was ever noted. In the female, this is understandable since the chancre may appear on the cervix or within the vagina where it is not easily discernible. In the male it is possible that the chancre occurs intra urethrally or it may be so insignificant and transient that it escapes notice.

Clinical Appearance—The typical hard chancre is an eroded indurated reddened *papule* or a coinlike lesion of cartilaginous consistency with a pink moist surface. Unfortunately the appearance may be wholly atypical and may assume many disguises. The chancre may appear as a *herpetic vesicle* without induration as a *ragged ulcerated lesion* simulating the chancroid (p 288) and as a *balanitis* or a *crusted sore*. It may occur in conjunction with a *scabetic eruption* on the penis. It may be conjointly present in a *chancroidal ulcer* or it may lurk as an inconspicuous erosion at the base of a *venereal wart* (p 3291). The chancre is usually single but multiple lesions occur especially in the Negro.

Sites of Lesions—The primary lesion occurs on the *genitals* in 93 to 95 per cent of all instances. It is *extragenital* in the remaining small percentage of cases where it occurs on the *lips* from kissing or the use of contaminated drinking glasses on the *fingers* in dentists physicians or midwives on the *tonsil tongue* or in the *perianal regions* in those who practice sexual perversions (p 2412). Chancres have also been noted on the *neck nipples chin* and *hands*. In the latter instance it is often due to human bites (p 3198).

The *extragenital primary lesion* is most often lacking in the typical characteristics. It is most commonly an *indurated papule* whose surface is eroded and crusted. On the *digit* or at the *nail margin* it may resemble paronychia (p 3973) although it is more indolent less painful and pus is absent except with secondary pyogenic infection. A characteristic feature of extragenital chancre is the prominence of the associated lymphadenopathy.

On the *tongue* the lesion may appear as an indurated superficial ulcer. On the *tonsil* there is a striking similarity to a *Vincent infection* (p 335). The tonsil is swollen indurated and there is peritonsillar redness and some yellowish membranous deposits. The *cervical chancre* may resemble a simple erosion or it may consist of an edematous bright red indurated and eroded area (Fig 959).

Lymphadenopathy—Associated with the genital lesion is a *bilateral inguinal lymphadenopathy* which develops about five to seven days after the appearance of the chancre. The nodes are usually painless but may be somewhat tender. The enlargement is moderate and may be greater on one side than the other. The nodes are firm rubbery discrete and not adherent to the skin. Suppuration does not occur unless there is a mixed infection, especially with chancroid.

Diagnosis—The diagnosis of the chancre rests upon the use of the *dark field examination* (p 45) for every suspicious lesion. The clinician who entertains a high index of suspicion relative to all anogenital eruptions chronic dermatoses of uncertain origin and ulcers associated with marked

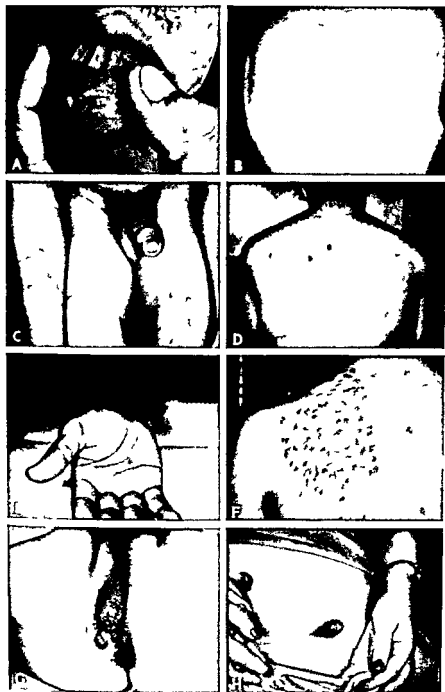


Fig. 960—Clinical manifestations of syphilis. Secondary and tertiary syphilis. A Mucous patch of lower lip. B Macular syphilid of body. C Varioliform syphilid of legs. D Erythema multiforme-like syphilid of back. E Papular syphilid of palms. F Maculopapular syphilid at site of mustard plaster. G Gumma of ulna. H Gumma of thigh.

Not even the expert will attempt a final differential diagnosis on morphological grounds. *Only by the dark field examination can a state of certainty exist.* The practitioner is urged to refer the patient with the suspicious lesion to the specialist who is particularly expert and experienced in the recognition of the *Treponema pallidum*.

SECONDARY SYPHILIS

The generalization of syphilis becomes manifest through the secondary lesions of the skin and mucous surfaces. Frequently these are associated with mild subjective symptoms such as *sore throat* and *articular pain*. *Generalized lymphadenopathy* may be marked.

Lesions of the skin may be present alone or they may be accompanied by manifestations involving the *mucous surfaces*. The eruption may be widespread and profuse or sparse and localized. The cutaneous manifestations are so varied and confusing that *dark field microscopy* and *serologic tests* are warranted in any eruptive phenomenon that is not absolutely pathognomonic of some characteristic dermatosis. Whereas the dark field examination leaves no doubt as to the etiology of the disturbance *serologic tests may be confusing*. They may be *negative* with the tertiary lesion. They may be *positive and unrelated to the dermatosis* as an acne vulgaris or a psoriasis occurring in a syphilitic individual. Finally they may be *misleading* as in the false positive reactions that occur in *infectious mononucleosis* (p 466) *leprosy* (p 273) and *vaccinia* (p 424).

The Macular Syphiloderma—The simplest and one of the most frequent rashes of secondary lues is the macular syphiloderm or *roseola syphilitica*. This is a pale pink or reddish macular rash seen best on the *lateral aspect of the flanks*. It may be profuse or sparse and it is easily overlooked unless sought. It is intensified by a hot bath and injection of an arsenical (Herxheimer reaction). It confines itself usually to the trunk.

The individual lesion is a pink rounded nonelevated blotch usually a centimeter or less in diameter. No scaling is present nor does any appear as the lesion involutes. The macular syphiloderm may have an associated papular quality and it is then called a *maculopapular eruption*.

The history, the presence of a healed or healing chancre, the morphology of the lesion, the positive serologic test of the blood and the absence of symptoms pertinent to other diseases aid in differentiation from *drug eruptions* (p 3335), the rose spots of *typhoid fever* (p 225), *pityriasis rosea* (p 3410), *rubella* (p 417) and *roseola* (p 409).

Papular Syphiloderma—The papular syphiloderm consists of infiltrated raised conical or flat coppery or reddish brown lesions. They vary from the size of a pinhead to that of a pea. The eruption is *widely dispersed* and may involve the *scalp face extremities and trunk*. Lesions are often present on the *palms and soles*. They may be fairly few in number or there may be a great profusion of papules.

Split Papules—Papules at times are present at the *corners of the mouth*. These develop a *horizontal fissure* because of the opening of the mouth and are known as *split papules*. They are to be differentiated from *perleche* (p 3302) and *ariboflavinosis* (p 624) by *dark field microscopy*.

Moist Papules (Condylomata Lata)—Papules assume a characteristic appearance when located on surfaces where *friction heat and moisture* prevail such as the *genitals* especially in females the neighboring areas the

| | |
|------------------------------|--|
| Rat Bite Fever | Maculopapular eruption of chest and arms after bite. Identifies spirochetes by darkfield after lymph node aspiration. (p. 365) |
| Rocky Mountain Spotted Fever | Acute febrile illness with lesions first on wrists and ankles later spreading to body. Weil-Felix positive (p. 372) |
| Rubella | Acute transitory maculopapular eruption usually in children (p. 417). Marked posterior cervical lymphadenopathy. No enanthem. Spreads from face to body and extremities. |
| Sarcoidosis | Brownish nodules or papules especially of face. Biopsy (p. 3271) |
| Syphilis | Macular or papular syphiloderms may be plantar and palmar. Darkfield and serology positive (p. 331) |
| Tinea Versicolor | Tan to brown scaling macules due to fungus. Alebrile |
| Tularemia | Acute febrile illness after handling rodent. Maculopapular eruption becoming pustular. History of tick bite. Positive blood culture, agglutins or skin test (p. 323) |
| Typhoid Fever | Roseola on abdomen in second week of illness. Fade on pressure. Positive blood or stool cultures and specific agglutination (p. 225) |
| Typhus Fever | Acute febrile disease with macules of axillae and loins later spreading to abdomen, chest and back. Weil-Felix test positive in serum (p. 372) |
| Urticaria Pigmentosa | Pigmented wheals which form wheal after rubbing. Alebrile. Biopsy (p. 3158) |
| Xeroderma Pigmentosum | Pigmented macule with areas of atrophy, telangiectases and keratoses on exposed parts after solar exposure. Alebrile. Precancerous. Biopsy (p. 3153) |

perianal region, the submammary folds and the axillae. These papules or *condylomata lata* become flattened, soft and grayish in color. At times they are distinctly hypertrophied and *papillomatous* resembling warts (p. 3288).

Mucous Patches—Papules may develop on the mucous surfaces of the lips, the buccal mucosa, the soft palate, tonsils or tongue. These appear as flattened barely elevated round or irregular grayish white mucous patches. Rarely the mucous patch may become superficially ulcerated. The moist lesions are notoriously infectious and teem with treponemes.

Mucous patches are to be distinguished from *herpetic* (aphthous) stomatitis (p. 1695), *Vincent's stomatitis* (p. 1698) and *drug eruptions* (p. 3335). The darkfield examination leaves no doubt in the differentiation.

Palmar and Plantar Syphiloderms—The palmar and plantar syphiloderm may exist as a part of the generalized papular syphiloderm. At times it is seen as the sole cutaneous manifestation of an active syphilitic infection. This eruption is extremely characteristic and almost pathognomonic. The lesions are discrete scaling macules or papules reddish to dark brown in color. The scaling is most marked at the periphery of the lesion forming a *collarette*.

DIFFERENTIAL DIAGNOSIS OF

Enanthems Involving Oral and Buccal Surfaces

Eruptions involving oral and buccal mucous membranes may represent local processes or manifestations of profound systemic disturbances. Recognition of the general characteristics of the individual lesion may enable dentist or practitioner to arrive at a correct diagnosis.

DIAGNOSTIC FEATURES

Erythema

Infectious stomatitis without other manifestations. Scarlet fever with exanthem and Schultz-Charlton blanching reaction. Secondary syphilis with positive darkfield findings and serology.

Vesicles and Bullae

Infectious stomatitis, aphthous stomatitis, herpes simplex and herpes zoster without manifestations elsewhere. Contact dermatitis from cosmetics or dentifrices. Dermatitis medicamentosa from drugs. Erythema multiforme, pemphigus and dermatitis herpetiformis with cutaneous dermatoses. Varicella and varicella with fever and exanthem. Foot and mouth disease with extremity lesions.

Macules and Papules

Fordyce's condition and leukoplakia without manifestations elsewhere. Lichen planus with exanthem. Measles with Koplik spot and exanthem.

Tumors

Epulis in pregnancy. Oropharyngeal cysts with x-ray changes and fluid by diagnostic puncture (p. 1714). Oropharyngeal neoplasms clarified by biopsy (p. 1656). Gummas with positive serology and therapeutic response to iodide.

Petechiae and Purpuras

Scurvy with therapeutic response to ascorbic acid. Vitamin K deficiency with therapeutic response to menadione. Leukemias and thrombocytopenic purpura with pathognomonic hemograms (p. 3704). Subacute bacterial endocarditis with positive blood culture.

Telangiectasis

Hereditary telangiectasis with similar lesions elsewhere and normal hemogram.

Ulcers

Aphthous stomatitis without lesions elsewhere. Scurvy with therapeutic response to ascorbic acid. Vitamin B deficiencies with therapeutic response to thiamine, riboflavin or niacin. Erythema multiforme, lupus erythematosus and dermatitis herpetiformis with cutaneous exanthem. Ulceromembranous stomatitis or gingivitis with Vincent organisms demonstrable on smears (p. 50). Epithelioma or carcinoma with positive biopsy. Tuberculosis with acid fast organisms demonstrable in exudate or tissue (p. 50). Diphtheria with membranous exudate and characteristic organisms in smears and cultures (p. 302). Primary or secondary syphilis with positive darkfield and/or serology. Tertiary syphilis with therapeutic response to iodide. Agranulocytosis and leukemia with characteristic (p. 3704). Typhoid fever with positive or elevated agglutination titer.

Pigmentations

Normal deposits in negroes and brunettes
 Nicotine tanning in habitual smokers Deposition of lead and bismuth with positive urine finding (p 3659) Adrenal cortical deficiency with hypotension and a thymia

Psoriasisform or Papulosquamous Syphiloderma—On occasions the papular syphiloderma becomes exceedingly *scaly* and the lesions have a close similarity to those of *psoriasis*. These eruptions have a tendency to group themselves over the *arms* and *upper trunk* but occasionally are more widely distributed. They are often seen as evidences of *relapse* in inadequately treated syphilis and may be stubborn and resistant to treatment.

Differentiation from *psoriasis* (p 3414) may not be easy since *psoriasis* may erupt in an individual with a positive serologic test of the blood. The absence of the characteristic nail changes of *psoriasis* and the distribution of the lesions will assist in differential diagnosis but at times histopathologic study may be required to render a final diagnosis.

Annulopapular Syphiloderma—An unusual eruption seen almost exclusively in Negroes is the annulopapular syphiloderma. This is a *ring shaped* lesion whose border is elevated and whose central portion is brownish to black and not at all elevated. The center is *hyperpigmented* at times almost black in color. The most common location is the *face* and around the mouth but lesions are at times present on the *genitals* and in rare instances more widely distributed on the trunk.

Follicular and Pustular Syphilodermas—Among the rarer types of secondary syphilitic eruptions are the follicular and pustular syphilodermas. The follicular syphiloderma consists of grouped pinhead sized papules located at the openings of the hair follicles and distributed largely on the trunk. It is seen most frequently in colored females and resembles *lichen scrofulosorum* (p 3269) and the *keratosis pilaris* of vitamin A deficiency (p 3236).

The *pustular* or *rupial* syphiloderma is quite rare and seems to afflict individuals whose general health is poor because of associated alcoholism and malnutrition. The lesion occurs as brownish crusted eroded or ulcerated moist and purulent areas varying in size from a pea to a quarter. The heaping up of the scales in strata is very typical. The rupial or pustular syphiloderma must be distinguished from *ecthyma* (p 3252) *impetigo contagiosa* (p 3251) and *drug eruptions* (bromides and iodides) (p 3335).

Pigmentary Syphiloderma—A rare manifestation of the secondary stage seen chiefly in women is the pigmentary syphiloderma or *leuoderma colli*. It consists of rounded pea sized or larger whitish *depigmented areas* seen mostly on the back of the neck but occasionally distributed over the *arms* and trunk.

Syphilitic Alopecia—Alopecia of a typical variety is observed in the secondary stage. It occurs on the scalp in irregular small patchy areas and is incomplete so that the resultant effect is described best as moth eaten. There may be concomitant alopecia of the eyebrows of similar appearance.

Varicelliform and Varioliform Syphilids—Secondary eruptions may bear a close resemblance to chickenpox or smallpox. The *varicelliform* and *varioliform* syphilids are encountered with great infrequency. Differential diag

nosis may be difficult since there may be associated systemic symptoms. The absence of the exanthematic diseases in epidemic form, a history of a previous attack of the exanthem, the age of the patient, a history of and concomitant signs of syphilis aid in reaching the final diagnosis. *Dark-field examinations* and *serologic studies* of the blood should be performed.

LATE (TERTIARY) SYPHILIS

The *gumma*, *nodulo ulcerative syphiloderms* and *leukoplakia* are the distinctive lesions of the skin in late syphilis. They may appear within four or five years after acquisition of the infection or as late as half a century after the chancre.

Tertiary syphiloderms differ from secondary lesions in many important characteristics. *Secondary syphiloderms* tend to be widely disseminated, generalized and symmetrical. They contain treponemas and are highly infectious. The *tertiary* or *late lesions* of syphilis are *localized*, *unilateral* and scant in number, and treponemas are rarely if ever demonstrable. They are relatively noninfectious and are considered a form of *allergic* response of a sensitized skin to a few organisms. The secondary lesions rarely leave scars, whereas the tertiary lesions do so regularly.

Gumma—The *gumma* may be *single* or *multiple* and exists as a rounded, elevated, pink or red *tumor*, usually painless. There is a gradual increase in size of the lesion, softening, fluctuation and finally breaking through of the skin with discharge of thick, purulent material and the formation of a punched-out, rounded *ulcer*. Favorite locations are the upper half of the *calf*, the *thigh*, *buttocks* and *trunk*. Involvement of the soft tissues of the tip of the *nose* is notorious for its resistance to therapy, although fever therapy may effect a cessation of activity.

The *gumma* may also appear on the tongue and on the soft and hard palates. *Gumma of the tongue* occurs as a solitary, rounded and firm mass, usually about the size of a small cherry. In time ulceration ensues and differentiation from *squamous cell epithelioma* (p. 3223) becomes vitally important. The presence of a known syphilitic infection does not preclude the diagnosis of malignancy and even some response to antisyphilitic therapy is unconvincing. It is deemed wisest to proceed with specific treatment but to perform a *biopsy*. *Gumma of the soft palate* produces a large, painful, necrotic ulceration. If treatment is not instituted early, considerable destruction of tissue may result.

Nodulo ulcerative Syphiloderma—The *nodulo ulcerative syphiloderm* is distinguished by its ability to produce varied *geometric patterns*, circles, segments of circles, kidney shaped and scalloped effects. The borders of these lesions are active and advance by centrifugal spread while the central portions consist of healed lesions which reveal themselves as thin, fine, yellowish, whitish or faintly brown areas of scarring. Unlike *lupus vulgaris*, the scarred area does not contain any signs of active disease. The border is composed of discrete, raised, nodular lesions separated by areas of scar where nodules have already healed. The nodules are generally *ulcerated*, the ulcers tending to be punched out or covered with an adherent yellowish crust. These lesions extend peripherally in an extremely slow manner and may remain active for many years. The *nodulo ulcerative lesion* may be single or multiple and is seen on any part of the body. It is to be differentiated chiefly from *lupus vulgaris* (p. 3262).

Leukoplakia—Also seen in the late stages of syphilis is leukoplakia of the *oral cavity* (p 1691) This lesion occurs as well in nonsyphilitic individuals especially heavy users of tobacco in any form It is seen as slightly raised whitish or grayish white adherent *plaques* on the *lips buccal mucosa* and *tongue* At times the patches undergo hypertrophic changes and become considerably raised and thickened They may develop *fissures nodules* and *ulcerations* In time degeneration into *squamous cell epithelioma* (p 3223) may take place

Elimination of all forms of tobacco removal of carious and sharp teeth good dental hygiene avoidance of very hot and irritating foods are excellent prophylactic procedures There are some who feel that the arsenicals should not be administered since they may stimulate epithelial hyperplasia and actually induce malignant transformation The local application of silver nitrate and similar caustics may excite epithelial overgrowth and should never be used

PRENATAL (CONGENITAL) SYPHILIS

Prenatal syphilis may become manifest wholly by the external evidences These are generally absent at birth but appear some time within the first few weeks of life

The patient may develop *syphiloderms* consisting of macular maculopapular and papular eruptions *condylomata lata* about the anus and genitals *fissures* about the mouth *dactylitis periostitis* and *osteomyelitis* snuffles with seropurulent or sanguineous nasal discharge and *mucous patches* in the mouth The characteristic *bullous eruption* seen only in prenatal syphilis consists of pea to coin sized bullae on infiltrated or eroded bases They are most frequent on the palms and soles though they also occur on the trunk and extremities

In *latent prenatal syphilis* when the diagnosis is not made early in life the child may develop other manifestations such as *interstitial keratitis* (p 1628) *gummatous or nodulo-ulcerative lesions* of the skin or mucous membranes *deafness* due to involvement of the auditory nerves or *disease of the central nervous system* (juvenile tabes or paresis) Other important late stigmata are the *Hutchinson teeth* affecting chiefly the upper central incisors and more rarely the lower The teeth are peg shaped (screw driver) thickened interoposteriorly and have a notch on the free border *Higoumenakis sign* is a unilateral thickening of the sternal end of the clavicle of the right side in right handed individuals and of the left side in the left handed *Clutton's joints* consist of bilateral hydrarthrosis of the knee joints and less commonly of the elbow joints The *saber tibia* is an anterior bowing and thickening of the shin bone due to osteoperiostitis Finally the patient may show a *saddle nose* due to gummatous destruction of the nasal septum

The *Hutchinson triad* consists of the Hutchinson teeth interstitial keratitis and deafness

MOLLUSCUM CONTAGIOSUM

Molluscum contagiosum (p 436) is an infectious disease of the skin characterized by the presence of one or more small pearly *umbilicated tumors* It occurs in both sexes and at all ages but is most common in children Infection may be acquired by direct contact or in schools asy

lums gymnasiums Turkish baths and swimming pools The disease is seen most frequently in the clinic

Etiology—The infective agent is a *filtrable virus* and the period of incubation may vary from weeks to months

The Lesion—Lesions may occur on any part of the body except perhaps the palms and soles They are common on the *face trunk* and about the male *genitals* There may be one or two lesions or as many as a hundred may be present They appear as pinhead to pea sized domed waxy or pearly *tumors* with a central *depression* (umbilication) There is no inflammatory component or halo except where secondary irritation or infection is present When the lesion is squeezed a cheesy material is expressed from the central depression Lesions may disappear spontaneously but since they are auto inoculable new ones keep presenting themselves

Pathology—Molluscum contagiosum has a distinctive histopathology The lesion is sharply demarcated from the healthy skin and lies completely within the epidermis It appears as a lobulated tumor which compresses and flattens the papillae and which is composed of altered epithelial cells These cells are large rounded whitish bodies with little or no nuclear structure They are present in the cheesy material expressed from the tumor and can be identified microscopically as the so called molluscum bodies

Diagnosis—Once seen it is difficult to confuse this condition with any other dermatosis At times distinction from warts and fibromas may be necessary *Warts* (p 3288) are most common on the hands unusual to rare on the body and frequently are papillary in structure The *fibroma* (p 3206) is rarely multiple usually larger in size deeper in the skin and lacks the waxy umbilicated appearance of the molluscum

Treatment—The lesions may be removed without leaving any scar This is accomplished by inserting a pointed scalpel slantwise beneath the lesion and levering it out bodily The same result is achieved by scraping the lesion away gently with a fine sharp curet After removal the base is touched with silver nitrate A simple method consists of nicking the lesion and squeezing out the contents completely It is important because of auto inoculability to rid the body of all lesions

WARTS (VERRUCAE)

Warts are benign epithelial growths of variable size differentiated by their shape and location Besides the *common type* (*verruca vulgaris*) the *juvenile flat* and *venereal warts* (*condyloma acuminatum*) occur The common wart may be *plantar filiform* or *digitate* These varieties will be described separately

Etiology—Filtrates of warts have been successfully inoculated into humans The incubation period is prolonged and may be one to eight months Warts can be "caught" but apparently not with equal facility by all Thinness of the skin may be a factor since warts are most common in children and young adults The oft repeated observation of a first or "mother" wart followed at a later date by younger warts in the immediate neighborhood is testimony to the *auto inoculability* of the virus Irritating genital discharges gonorrhea and poor bodily hygiene predispose to venereal warts

Pathology—Histologically the wart is a circumscribed epidermal lesion consisting of more or less *hyperkeratosis* and *acanthosis* (p 3101) No change is present in the corium except where there is secondary inflammation with cellular infiltration and vascular dilatation

Clinical Variants—Warts appear as the common juvenile and venereal varieties

The Common Wart—The common wart (*verruca vulgaris*) is a pin head to pea sized lesion skin-colored grayish or brownish in color firm and presenting an irregular cauliflower like (warty or verrucous) surface. At times the verrucous appearance is difficult to detect except with a lens the surface seeming smooth and regular. The common wart is most

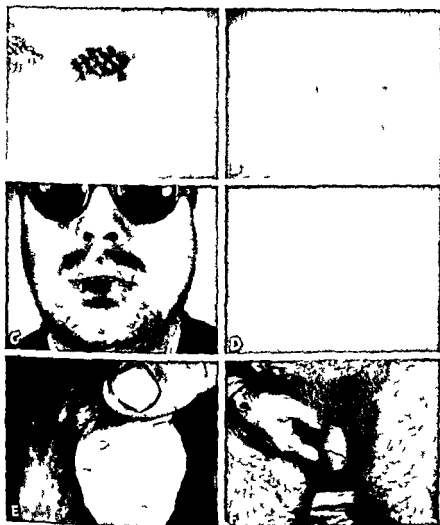


Fig. 961.—Herpetiform lesions. *A* Herpes zoster of shoulder in vesicular stage. *B* Herpes zoster of shoulder in gangrenous stage. *C* Herpes simplex of lip. *D* Molluscum contagiosum generalized. *E* Molluscum contagiosum of glans penis. *F* Herpes simplex of penis suggesting venereal lesion.

frequently observed on the *fingers* and *hands* but is also seen beneath the *finger-nails* and on the *scalp* and *face*. It may occur as a single or multiple lesion.

The *plantar wart* is a special variety of the common wart occurring on the *heel* or *ball* of the foot and less often on the plantar surface of

 DIFFERENTIAL DIAGNOSIS OF THE

Commoner Generalized Vesicular and Pustular Eruptions

Other than chickenpox and smallpox generalized vesicular and pustular eruptions are uncommonly encountered in clinical medicine. It is only rarely that syphilis, impetigo, herpes or contact dermatitis spreads to involve the entire body. Except in syphilis the laboratory gives minimum assistance under these conditions and the practitioner is dependent almost wholly upon his clinical acumen.

DIAGNOSTIC FEATURES

| | |
|-----------------------------------|--|
| Chickenpox | Acute infection with pleomorphic monolocular vesicles first seen on body (p. 420) |
| Dermatitis Herpetiformis | Rare symmetrical dermatosis with grouped papules, vesicles, bullae and pigmentation. (p. 3371) |
| Dermatitis Medicamentosa | Pustules particularly after iodide, bromide and antimony (p. 3335) |
| Dermatitis Venenata | Vesicular eruption with intense itching from contact substances especially poison ivy (p. 3330) |
| Dermatitis Exfoliativa Neonatorum | Generalized form of impetigo contagiosa in newborn (p. 3252) |
| Dermatophytids | Vesicular dermatophytids may accompany ringworm of hands and feet (p. 3299) |
| Erythema Multiforme | Widespread skin and mucosal lesions may be vesicular as well as macular and papular (p. 3375) |
| Foot and Mouth Disease | Vesicular eruption of virus origin. Involves mouth, hands and feet (p. 437) |
| Generalized Vaccinia | Vesiculopustular eruption associated with inoculation of cowpox vaccine (p. 428) |
| Glanders | Generalized pustular eruption containing chocolate-colored pus in persons handling horses. Get skin test and culture for <i>M. mallei</i> (p. 327) |
| Herpes | Usually localized vesicular eruption in simplex and zoster varieties. Rarely generalized as in herpes gestationis in pregnancy (p. 433) |
| Hydroa Estivale | Vesicular and bullous lesions in children sensitive to sunlight |
| Keratosis Blennorrhagica | Vesiculopapular eruption with gonorrheal infection (p. 3257) |
| Miliaria | Inflammatory vesicles and papules with itching |
| Molluscum Contagiosum | Umbilicated vesicles of virus origin. Predilection for face, trunk and genitals (p. 3287) |
| Pemphigus | In early stages eruption may be vesicular before formation of bullae (p. 3405) |
| Smallpox | Acute infection with monomorphic polylocular vesicles and pustules spreading from face to body (p. 424) |
| Solar Dermatitis | Sunburn (p. 3140) |
| Staphylococemia | Generalized furunculosis with bacteremia (p. 153) |

Sudamen

Multiple non-inflammatory crystalline vesicles in febrile or sweaty patients

Syphilis

Secondary eruption may be variceliform or varioliform (p. 338)

the toes. Here the verrucous appearance is rarely observed; the projections being flattened by pressure. The plantar wart resembles a callosity, but close inspection and paring will reveal the presence of multiple verrucous centers which have become agglomerated. Pain is more marked in this location. One or more such lesions may be present.

The *filiform wart* is encountered most often on the neck, the face and the eyelids. It consists of a single thread-like projection, usually less than $\frac{1}{2}$ inch in length, soft and freely movable. This type is usually multiple.

The *digitate wart* is seen practically exclusively on the scalp. It is composed of a closely packed mass of thread-like projections. These projections are separated one from the other almost down to the scalp. The lesion usually attains the size of a pea and bleeds easily when bruised as in combing the hair. Usually one finds no more than one or two such lesions present.

The Juvenile Wart—The juvenile flat wart is a flat, smooth, slightly elevated papule, skin-colored, tan or brownish and rounded or irregular in outline. It is practically always multiple and may be extremely numerous. Linear arrangement of the papules along the lines of scratch marks is at times noticed. The sites of predilection are the backs of the hands, the forearms and the face.

This wart may be mistaken for *lichen planus* (p. 3389) but it lacks the violaceous hue, the shining surface, the pruritus and the usual distribution of the latter condition.

The Venereal Wart—The venereal wart (*condyloma acuminatum*) is usually multiple and consists of pointed or rounded, sessile masses, pink, dark red or dirty gray in color. At times it forms projections resembling a raspberry or becomes a hypertrophic, thick, fleshy, irregular tumor. It is often bathed in secretion which emits a foul odor. In the male, it is seen most commonly on the glans penis, in the coronal sulcus and on the inner surface of the prepuce; in females, on the labia. The perianal region is a fairly common location. Lesions may be present within the vagina and on the anal mucosa. If these are not extirpated, they serve as a constant source of reinfection for the skin. A thorough inspection of the vaginal and anorectal mucosa is necessary before treatment is begun.

The venereal wart must be differentiated from the moist papule of *secondary syphilis* (p. 3281). The latter is a flat lesion without any verrucous appearance and tends less to form large masses. Other concomitant evidences of secondary syphilis are frequently present; the darkfield usually reveals the *Treponema pallidum* and the serologic tests of the blood are always positive.

Treatment—The treatment of the wart depends upon the type and location of the lesion under consideration. The quickest and most effective method for the removal of warts is *curettage*. This must be performed under local anesthesia; an adequate amount of 2 per cent procaine solution being injected beneath the lesion. The wart is then thoroughly scraped

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CHAPTER 148

THE SKIN IN INFECTION SUPERFICIAL AND DEEP DERMATOPHYTOSIS AND DERMATOMYCOSES CUTANEOUS LEISHMANIASIS AND HELMINTHIASIS

SUPERFICIAL DERMATOPHYTOSIS AND DERMATOMYCOSES

The superficial fungous infections are caused by *epidermophytons trichophytons* (including *achorion*) *microsporon*s and *yeasts*. These fungi have an affinity for keratin and thrive best in the stratum corneum and in the hair and nails. They do not invade the internal tissues or organs. Optimal conditions for their growth and multiplication are provided by keratin, the normal body temperature, the presence of moisture and acid sweat.

The clinical syndrome is usually distinctive but in many instances the diagnosis of a fungous infection necessitates laboratory study and other investigations. The simplest and most useful laboratory procedures are the *direct examination* of scrapings, *cultural studies* and tests of specific *cutaneous sensitivity*. The lesion is also observed under *filtered ultra violet light* (p. 3794).

RINGWORM OF THE BODY (TINEA CORPORIS TINEA CIRCINATA)

Ringworm occurs on the face, neck, body and extremities. Lesions may be single, multiple or innumerable. Children are more commonly affected but the disease is seen also in adults. It may be acquired from other affected persons or from animals, particularly cats and dogs. The most common fungi involved are those of the *Microsporon* or *Trichophyton* families.

Clinical Appearance—Primary ringworm infection of the skin produces mainly a *dry scaling lesion* or a *moist crusted disturbance* of the nonhairly skin. Variants occur but these forms comprise the mass of cases observed.

The *dry scaling type* consists of rounded or ovate lesions whose center is clear or clearing so that the *ring shape* is suggested. The border is slightly elevated, reddened, scaly and contains some minute vesicles. Such lesions may be very small or they may enlarge to the size of a palm.

The *moist crusted variety* shows a much more marked *vesicular response* with less tendency to central clearing and ring formation. Considerable oozing and actual crust formation may be present (Fig. 962-1).

Diagnosis—Differential diagnosis may require the ultimate and incontrovertible proof of the presence of fungi by direct examination and cultural techniques. *Itchyiasis rosea* (p. 3410) may be simulated, especially when the lesions are numerous. *Itchyiasis rosea* is usually more widespread and has a rather characteristic pattern of distribution and arrangement; the lesions rarely show vesicles and the center of the lesion is not clear.

The crusted vesicular variety may simulate *contact dermatitis* or *impetigo contagiosa*. *Contact dermatitis* (p. 3330) is usually more pruritic, more inflammatory, more resistant to therapy and contains no fungi. *Im*

away removing all the diseased tissue with a sharp skin curet. Some prefer to destroy the verrucous tissue with the *electrodesiccating needle* before curetting. After the lesion has been ablated, bleeding is controlled by pressure, the base is desiccated, or silver nitrate is applied. A dressing of sterile gauze with Boric Acid Ointment is used and changed daily or on alternate days.

Warts may be destroyed with the *electrodesiccating needle* but this has the disadvantages of uncertainty as to depth of destruction and delayed healing (electrical burn). Local anesthesia is necessary in this method also. It is poor procedure to desiccate a wart and allow the necrotic tissue to remain as this frequently leads to secondary infection. All destroyed tissue should be removed after desiccation and boric acid ointment dressing applied.

The use of *roentgen rays* and *radium* for the treatment of warts is a procedure only for those who possess specialized training. As a rule large doses are required and while the method is painless and nondisabling it involves a certain amount of risk and should be reserved for plantar warts and those occurring in diabetics, elderly persons and those with a bleeding tendency.

A simple technic is the repeated application of *caustic chemicals* such as *trichloroacetic acid*, *silver nitrate*, *glacial acetic acid* and pure *phenol*. The disadvantages are the necessity for several applications and the dangers of chemical burn to healthy tissues. A layer of petrolatum should be placed around the wart to protect healthy skin and then the chemical thoroughly applied. After the destroyed portion of the lesion has separated another treatment may be given.

The juvenile flat wart may be treated with methods of suggestion (p. 1316). Other procedures include painting with *dyes* (gentian violet, mercurochrome), the momentary application of the lightest possible *desiccating spark* or minute doses of *roentgen rays*, the use of *desquamating lotions* (lotio alba), *desquamating doses of ultraviolet light* or *cold quartz*, the oral administration of *Fowler's Solution* or *Protiodide of Mercury* $\frac{1}{8}$ grain two to four times daily.

With venereal warts good results also may be obtained with one or two applications of podophyllin in mineral oil.

petigo contagiosa (p 3251) has a tendency to produce larger vesicles or flaccid bullae with copious contents the crusts stuck on and no fungi are demonstrable

Treatment—See p 3307

RINGWORM OF THE GROIN (TINEA CRURIS ECZEMA CRURIS DHOBI ITCH)

The characteristic localization of *tinea cruris* sets it apart from other examples of ringworm of the smooth skin The causative organism is most commonly the *Epidermophyton inguinale* although others are at times responsible The condition is infectious and may be acquired by contact or the use of contaminated towels or clothing It is most often seen in adults more in males than females

Clinical Appearance—The eruption is sharply delimited from the normal skin and may consist of several discrete patches In its classical form it is composed of a single large continuous patch affecting the groins by



Fig 963—Ringworm of the groin (*tinea cruris*) Dhobie itch

laterally The eruption may spread upward to the pubis onto the scrotum and along the perineum to the sacrum Isolated patches may also be found at the umbilicus below the breasts in females and notably in the axillae The rash is pink to dark red in color very superficial scaly and its advancing border contains tiny vesicles It is usually not very inflammatory but the action of heat moisture and friction induce secondary inflammatory changes (eczematization) leading to variable amounts of pain burning and itching

Diagnosis—Differential diagnosis is usually simple *Erythrasma* (p 3301) is less inflammatory and the causative organism is very minute and can be seen best if at all under the oil immersion lens *Monal infections* (p 3301) generally show scattered *vesiculopustular* lesions outside of the border of the large intertriginous patch In *psoriasis* (p 3414) there is more

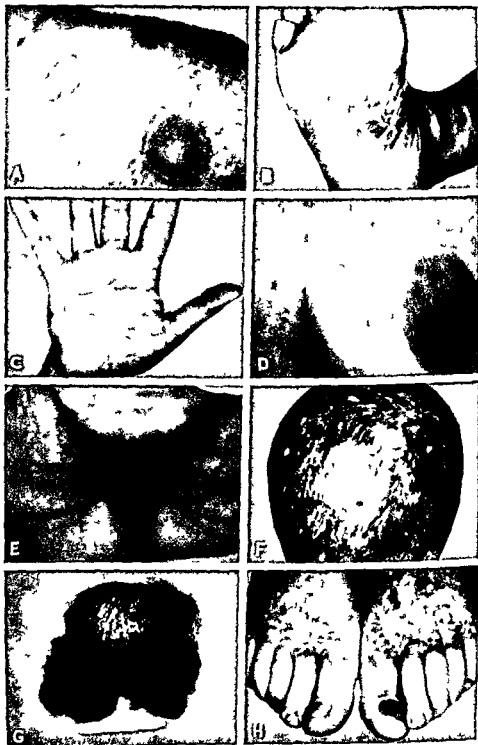


Fig 96.—A Ringworm of the body (*tinea corporis*) B Ringworm of the foot (*tinea pedis*) C Dermatomycosis (*erythema multiforme*-like) D *Tinea versicolor* E Moniliasis in a diabetic F Ringworm of the scalp (*tinea capitis*) G Favus H Ringworm of the nails (*onychomycosis*)

| | |
|-----------------------------------|--|
| Gout | Podagra involving great toe. Note redness and exquisite pain. Specific relief from colchicine. |
| Granuloma annulare | Dermatosis of children and young adults. Characterized by circinate nodules and papules of fingers and hands, elbows and toes. Get biopsy (p. 3384). |
| Granuloma pyogenicum | Indolent, proud flesh surrounding chronic paronychia (p. 3973). |
| Intertrigo | Erythema and maceration between fingers and toes. Usually complicated by fungus or monilial invasion. Seek offending pathogen by smear and culture (p. 3161). |
| Keratosis | Thickened scaling papules and nodules. Associated with advancing years, seborrhea, occupational exposure or arsenotherapy. A precancerous. Get biopsy (p. 3166). |
| Keratosis blennorrhagica | Vesiculopapular eruption especially of feet. Associated with gonorrheal invasion (p. 3257). Treat with penicillin. |
| Keratoderma climactericum | Fissures, keratosis and severe pain of palms and soles. Associated with menopause. Note therapeutic response to estrogen (p. 3240). |
| Keratoderma palmaris et plantaris | Rare dermatosis. With thickening of palms and soles, dryness, yellow coloration and diminished flexibility. Most often observed in Mediterranean countries. |
| Maduroomycosis | Fungous infection of foot. With edema, nodule formation, foul smelling discharge through multiple sinuses. Identify pathogen by smear or culture (p. 3315). |
| Melanocarcinoma | Pigmented neoplasm. Highly invasive by satellite extension or through lymphogenous dissemination. Get biopsy (p. 3225). |
| Moles | Pigmented and non-pigmented, hairy and non-hairy nevi (p. 3204). |
| Moniliasis | Yeast infection with vesiculation and pustulation. Usually interdigital. Demonstrate pathogen by smear or culture (p. 3301). |
| Paronychia | Pyogenic infection of tissues surrounding nail. |
| Pellagra | Vitamin deficiency causing redness, edema, desquamation and hyperpigmentation of exposed parts. Often associated with glossitis, neuritis and gastrointestinal manifestations. Note therapeutic response to nicotinic acid (p. 3237). |
| Perforating ulcers | Usually of toe. In association with peripheral vascular disease or neuropathies such as tabes dorsalis and syringomyelia. Make complete survey for delineation of underlying cause (p. 3228). |
| Pinta | A treponematosis producing keratosis, pigmented and depigmented areas of extremities including palms and soles. Supplement serologic examination with darkfield microscopy. Differentiate from syphilis by more rapid response to penicillin (p. 353). |

DIFFERENTIAL DIAGNOSIS OF

Dermatoses of the Hands and Feet, Fingers and Toes

See also Arms and Legs p 3378

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|------------------------|--|
| Acrodynia | Redness swelling and pain of hands and feet. A neonatal disturbance appearing in first two years of life. Note therapeutic response to vitamin B (p 3145) |
| Acroparesthesia | Tingling and other abnormal sensations of hands and feet. In elderly women (p 3230). Try estrogen therapy |
| Acrosclerosis | Scleroderma like changes. Usually seen in women with Raynaud's disease (p 3367) |
| Ainhum | Tropical disease resulting in amputations of fingers and toes (p 3370) |
| Callositas | Calluses of hands and feet. May be occupational or associated with errors in foot mechanics and shoeing |
| Chapping | Superficial fissures due to cold |
| Chilblains | Redness pain and swelling of the fingers and toes followed by vesiculation and gangrene. Due to prolonged exposure to excessive cold. Consider use of anticoagulants |
| Clavus | Hard and soft corns. May be exceedingly painful on plantar surface. If keratolytics fail consider x ray treatment |
| Contact dermatitis | Usually occupational but may also be caused by dyes polish nylon hosiery leg make up socks soap or nail dye. Verify with patch test (p 3330) |
| Decubitus ulcers | Over heels in bed ridden patients |
| Dermatophytosis | Ringworm of fingers and toes. With itching scaling maceration fissuring and denudation of the skin. Identify fungus by smear or culture (p 3293) |
| Dermatophytids | Vesicular lesions with intense itching. Most often seen on fingers and instep. Look for invading fungus elsewhere. Confirm with skin test (p 3299) |
| Epithelioma | Squamous cell malignancies. Occasionally seen on hands. Note raised border and chronic ulceration. Get biopsy (p 3220) |
| Erythromelalgia | Vasomotor neurosis. With episodes of violent pain, redness and swelling of the extremities. Most often seen in the female (p 1002) |
| Erysipeloid | Chronic erythema of the hands and fingers in meat handlers (p 3273) |
| Foot and mouth disease | Epizootic virus infection. Vesicular eruption of mouth hand and feet (p 437) |
| Ganglion | Rounded non inflammatory tumor of tendon sheath. Most often seen at wrist. Evacuate by pressure or excise |
| Glomus tumor | Exquisitely tender nodular growth. Often seen beneath nail bed (p 3207). Treat by excision |

be so trivial that the patient is entirely unaware of the infection it can assume severe and disabling proportions. Its treatment by a great many advertised proprietary medications often leads to secondary contact dermatitis which may produce a confusing clinical picture.

The responsible organisms most often are *Trichophyton gypsum* and *Trichophyton purpureum*. The latter produces a more chronic and resistant type of the disease. Both sexes are affected, males more often than females. The condition is contagious and the fungi may remain viable for long periods of time in fiber and rubber mats, wooden floors and articles of apparel. Public places where the bare feet are exposed such as gymnasiums, swimming pools and shower baths may act as sources of spread of the infection. Excessive sweating, heat and friction may act as predisposing factors. There is apparently a difference in susceptibility in different persons to the infection and certainly a marked variance in the reaction produced by the same fungus.

The Dermatophytoses—In its most minor form, *tinea pedis* may consist of no more than excessive *scaliness* and some *maceration* of the skin between the fourth and fifth toes. As the condition progresses there appear *vesicles* on the toes and soles and increased maceration and fissuring between the toes. Heat and moisture and friction produce an *acute inflammatory reaction* with edema, marked vesicle and bulla formation and raw eroded surfaces. *Secondary pyogenic infections* occur in these areas. Pruritus, burning and pain may be so great that the patient is unable to walk without great suffering.

Many individuals have the infection for years without symptoms, spread or exacerbation. Others have tendency to recurrent exacerbations in the summer. In some the disease confines itself to the toes while in others there are patches on the dorsa of the feet, the legs and thighs. These patches resemble the circumscribed inflammatory, eczema-like patches of *tinea circinata*.

The Dermatophytids—Associated with the dermatophytosis of the feet there is a frequent eruption of the *hands* which varies from a few insignificant and transient *vesicles* on the lateral surfaces of the digits to wide spread moist oozing vesicular inflammatory areas involving the digits and the dorsal and palmar surfaces. These hand eruptions are generally sterile. Fungi are not present in direct examination or culture. Because their relationship to the primary fungous infection of the feet is indubitable these hand lesions are considered *allergic manifestations* or *dermatophytids* produced by hematogenous dissemination of the fungi from the feet. There are however true instances of actual primary fungous infection of the hands.

The mechanism by which the dermatophytid is produced is of importance in the therapeutic approach. The fungi are carried in the blood stream to the site of predilection which has previously been sensitized. The organisms are destroyed but cutaneous lesions are produced. Viable pathogenic fungi are demonstrable in the lesions of the feet and in the blood stream but not in the hand lesions. A state of hypersensitivity of the skin is shown with fungous extracts (*trichophytin*) and most important of all the hand lesions tend to recur unless the primary focus is eradicated.

Isolated eczematous patches in which fungi cannot be found are also

| | |
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| Pityriasis rubra pilaris | Rare chronic inflammatory dermatosis characterized by horny papules surrounding hair follicles. Note nutmeg feel. Look for black core papules on dorsum of hand. Try therapeutic effect of large doses of vitamin A (p 3412) |
| Pyoderms | Localized abscesses. Usually caused by staphylococcal or streptococcal infections. Treat locally or systemically with antibiotics (p 3248) |
| Radiodermatitis | Atrophic keratotic and pigmentary changes in the hands of physicians and roentgenologists. A precancerosis (p 3160) |
| Scabies | Infestation with <i>acarus</i> . Note burrows and intense itching of hands and feet particularly in web areas. Identify parasite in lesion (p 3180) |
| Scleroderma | Chronic systemic disease producing diffuse induration and hidebound cutaneous covering. Eventually fatal (p 3427) |
| Synovial cysts | Bluish white tumors of terminal interphalangeal joints. Often seen in conjunction with hypertrophic osteoarthrosis. Puncture yields clear thick, gelatinous fluid (p 3208) |
| Syphilis | Particularly note palmar and plantar syphiloderms. Supplement serologic examination with darkfield microscopy. Note syphilitic dactylitis in neonatal infections (p 3285) |
| Tuberculosis verrucosa cutis | Circumscribed, indolent wartlike lesions of the dorsum of the hand. Usually seen in pathologists. Get biopsy (p 3259) |
| Tularemia | Primary lesion often on fingers. With satellite lymphadenopathy. Get history of bite of rodent. Attempt to isolate pathogen by smear or culture (p 323) |
| Uncinaria dermatitis | Papules, vesicles and bullae of soles of feet where hookworm ova have penetrated. Attempt identification of pathogen (p 1903) |
| Verrucae | Warts of various types (p 3288) |
| Xanthomas | Yellow papules, nodules and diffuse infiltrations of hands and feet. In association with diabetes mellitus and systemic reticulo-endothelioses. Get blood sugar and cholesterol levels. Conduct complete physical examination (p 3243) |
| Xeroderma pigmentosum | Congenital dermatosis. Seen in first years of life. Characterized by pigmented macules, white atrophic spots, telangiectases and keratoses of exposed areas. A precancerosis (p 3158) |

infiltration and scaling of the lesions with confirmatory typical psoriatic patches elsewhere on the body.

Treatment—See p 3307

RINGWORM OF THE FEET (TINEA PEDIS, DERMATOPHYTOSIS PEDIS, ATHLETE'S FOOT)

Ringworm of the feet occurs in 15 to 90 per cent of young adults but is seen also in children and older persons. While its manifestations may

of the primary disease rather than that of *tinea versicolor*. Of course fungi will be absent in the lesions *Syphilitic leukoderma* (*leukoderma colli*) (p 3285) most commonly affects females and the spots are seen on the back and sides of the neck although in exceptional instances they may extend to the trunk and upper extremities. Associated lesions of syphilis a positive serologic test of the blood and absence of fungi will assist in making the correct diagnosis. *Maculo anesthetic leprosy* (p 273) may be accompanied by achromic areas but there are usually other lesions of the disease and the depigmented areas are often anesthetic.

Treatment.—See p 3307

ERYTHRASMA

Erythrasma is a relatively uncommon superficial fungous infection which affects the *intertriginous regions* such as the axillae, genitocrural area and intergluteal cleft. It occurs as fairly large well demarcated patches of a tan or faintly brown color with slight superficial scaliness. The border may be somewhat reddish but does not contain vesicles.

The causative agent is the *Microsporon minutissimum* which can be seen best under the oil immersion lens. It is extremely difficult to grow on artificial culture media. The disease is seen in adults more often in males.

It must be differentiated from *tinea cruris* (p 3295) and *tinea versicolor* (p 3300). The former usually is more inflammatory and has a distinctly elevated and vesicular border. The latter has little tendency to remain localized in the intertriginous areas and consists of smaller discrete macules without any inflammatory border. Mycologic studies by direct examination and cultural methods definitely determine the diagnosis.

Treatment.—See p 3307

MONILIASIS

Infection of the skin and mucous membranes by a pathogenic yeast *Monilia albicans* may produce a variety of clinical syndromes. While many saprophytic yeast organisms are found on normal skin the *Monilia albicans* is definitely pathological. Monilial infections are most frequent in adults of both sexes. Diabetes mellitus, obesity, hyperhidrosis and prolonged contact with water act as *predisposing factors* to infection.

Clinical Manifestations.—Moniasis may present itself in various distinctive forms each of which will be separately described.

The *intertriginous forms* of moniasis resemble a simple intertrigo or *tinea cruris*. The monilial form is distinguished clinically by its tendency to display isolated vesiculopustular lesions outside the border of the intertriginous patch. It occurs in the axillae, inframammary clefts, anogenital area, umbilicus and the interdigital spaces of the feet. It can simulate the nonparasitic forms of intertrigo, *tinea cruris* and dermatophytosis of the feet. Differentiation is of more than academic value since the response to therapy differs. The intertriginous lesions are apt to be more edematous, inflammatory and moist in moniasis and have a scalloped edge with satellite vesiculopustules outside this edge. Monilial infections in the *perianal region* give rise to an intractable pruritus ani (p 3415). *Erosio interdigitalis blastomycetica* occurs in the web spaces of the hands. This lesion

seen on the feet legs and arms. These resemble the lesions of *tinea circinata* and are believed also to be dermatophytids.

Erysipelatous Dermatophytid—The eruption of erysipelatous dermatophytid is thought to be an allergic fungous manifestation. Its response to sulfanilamide would suggest that it is a form of streptococcic infection in which the organisms have gained access to the deep tissues of the leg through macerated and fissured areas of fungous infection in the web spaces.

Complications—The dermatophytoses often afford a portal of entry for the invasion by pathogenic bacteria. *Local abscess cellulitis*, and especially *lymphangitis* and *lymphadenitis* involving the glands of the groin are frequently produced. Erysipelas like manifestations are also observed with altogether too great frequency. In point of fact any infection involving the toes lower feet or the inguinal glands may be superimposed upon a fungous lesion that at times seems inconsequential.

Treatment—See p. 3307.

TINEA VERSICOLOR (PITYRIASIS VERSICOLOR CHROMOPHYTOSIS)

Tinea versicolor is a fairly common superficial fungus infection. The causative organism is the *Microsporon (Malassezia) furfur*, an organism which cannot be grown in artificial culture media. It occurs in both sexes and is most frequent in young adults.

The Lesion—The lesions of chromophytosis appear as a few to a vast number of small minutely elevated *macules*. These are finely *scaly* and vary in color from faint tan to brown. The usual distribution of the eruption is the *chest shoulders arms back and abdomen*. Extensive eruptions may extend to the scalp neck face and thighs (Fig. 962 D).

Achromia Parasitaria—When the affected skin is exposed to sunlight or ultraviolet radiation the typical lesions of the disease may disappear. The surrounding normal skin becomes tanned but the filtering or screening action of the lesions does not allow penetration of the ultraviolet rays and the affected areas do not become pigmented. The desquamating action causes the macule to scale off and the underlying skin now appears white by contrast with the sunburned normal skin. This effect has been termed *pseudo achromia* or *achromia parasitaria*. It is not a true achromia in the sense of an absence of pigment but merely a relative deficiency of pigment in comparison with the circumambient hyperpigmented skin. Further exposures to sunlight or ultraviolet irradiation increase the contrast since the pale areas do not tan well. The patient may only notice the condition for the first time after the appearance of these white spots in the summer.

Diagnosis—The diagnosis of the characteristic *tinea* with its noninflammatory tan or brown finely scaling macules is simple. *Pseudo-achromia* may need differentiation from *vittiligo*, *leukoderma*, *syphilitic leukoderma* and *maculo-anesthetic leprosy*. In *vittiligo* (p. 3404) the patches of depigmentation are larger more irregular and there is distinct zone of hyperpigmentation at their border. The condition is seen most often on the face hands and anogenital region. Scaling is absent and fungi are not found in scrapings. *Leukoderma* (p. 3404) is a relative and temporary depigmentation seen after the disappearance of certain cutaneous lesions especially in psoriasis. The presence of active lesions of the primary disease or a history of their presence suggests the diagnosis and the distribution is that

partial alopecia with little or no evidence of inflammatory reaction and without vesicle or pustule formation. Inspection of these areas reveals the presence of *broken off hairs* and some *scaling*. In certain instances there occurs a marked inflammatory reaction and the affected areas are edematous, reddened and tender. There may be a mucopurulent discharge which leads to crust formation. This condition is called *kerion* (Celsi) and may be associated with a widespread, even *generalized eruption* of tiny *papules* at the mouths of the hair follicles. These are allergic manifestations, a result of the dispersion of the fungi through the blood stream from the primary focus on the scalp. They are similar in pathogenesis to the dermatophytids of ringworm of the foot (p. 3298). These secondary allergic eruptions require little or no therapy, since they disappear spontaneously when the scalp lesion is controlled. In these cases the intradermal test with the fungous extract is strongly positive.



Fig. 901.—Ringworm of the beard (*tinea barbae*). Before (left) after fractional dose of filtered x rays (3 mm. Al.) without epilation.

Diagnosis.—The diagnosis of *tinea capitis* on the basis of clinical appearance is usually simple. It is readily confirmed by direct examination and culture. Filtered ultraviolet light (Wood's light) makes diagnosis immediate and reveals the extent of the involvement.

The usual variety must be differentiated from alopecia areata and the rare monilethrix. In *alopecia areata* (p. 3445) the loss of hair is more or less abrupt in circumscribed coin shaped areas and is without any inflammatory reaction or broken-off hairs. *Monilethrix* (p. 3440) is a congenital familial disease in which partial diffuse and not circumscribed alopecia occurs and the hairs present have a beaded appearance. The alopecia of secondary syphilis is usually also present in the eyebrows; other cutaneous lesions are also in evidence and the serologic tests of the blood are positive. *Kerion* differs from *furuncle* and *carbuncle* in the absence of pain and tenderness and the lack of free pus formation.

Treatment.—See p. 3307.

is rather sharply circumscribed purplish and has a moist and at times macerated surface

Perleche affects the commissures of the mouth and may be unilateral or bilateral. It has a moist reddened surface covered with a layer of macerated skin and at times fissuring is present. *Perleche* must be distinguished from the *syphilitic split papule* (p 3281) in which darkfield examination practically always reveals *Treponema pallidum*. Lesions suggestive of *perleche* have also been described in *arboflavimosis* (p 3238) but associated with them are *cheilosis* the sharkskin like eruption in the nasolabial folds and often a *glossitis*.

Mucosal infections with *monilia* are seen with a fair degree of frequency. *Thrush* (p 1697) occurs in infants and consists of greyish white membranous plaques involving the tongue buccal mucosa and palate. These membranes can be detached without great difficulty. *Vaginitis* (p 2597) due to monilial infection is accompanied by a more or less profuse discharge. This may act as a source of infection to the skin and accounts for the difficulty in curing *genitocrural moniliasis* in females.

Infections of the *nails* and *periungual tissues* give rise to a *chronic paronychia* due to *Monilia albicans*. The periungual tissues are thickened edematous and reddened. There may be intermittent but insignificant amounts of purulent discharge. Very little pain or tenderness is present but the condition is unsightly and is seen mostly in housewives and others whose hands suffer prolonged immersion in water. Associated with this may be other lesions of *moniliasis* as described above. Usually along with the *paronychia* the nail shows ridging becomes misshapen discolored and thickened.

Treatment—See p 3307

TINEA CAPITIS (RINGWORM OF THE SCALP)

Tinea capitis is an infection of the hair follicles and hairs of the scalp by various fungi. Ringworm of the scalp is seen almost exclusively in children before the age of puberty. It may be acquired from contact with infected individuals and endemic outbreaks appear in schools orphanages and wherever children associate closely. Infection from cats and dogs is not uncommon and towels hats and toilet articles may spread the *tinea*.

The predominant causative organisms are the *Microsporon lanosum* (animal origin) and the *Microsporon audouinii* (human origin) but occasionally examples due to *Trichophyton*s are encountered. Fungi of animal origin (*M. lanosum* and certain *trichophyton*s) are more apt to induce an inflammatory type of ringworm especially *kerion* while those of human origin (*M. audouinii* and occasional *trichophyton*s) rarely do. The difference is of great importance from the therapeutic and prognostic standpoint. While the presence of the fungus is readily demonstrated by direct examination of an infected hair (p 40) cultural methods are needed to determine the exact nature of the fungus. In the presence of *kerion fungi* may not be found because the violence of the inflammation often leads to their destruction. Intradermal tests with extracts of fungi or *trichophyton* may also be utilized.

Clinical Appearance The clinical picture of ringworm of the scalp is very characteristic consisting of one or more rounded greyish patches of

deals with disease caused by the *trichophytons* especially *T. gypsum* and *purpureum*

Clinical Appearance—The nails usually become distorted and irregular thickened or crumbly discolored yellow to brown and lose their normal luster The disease begins at the distal border or free edge of the nail and gradually progresses toward the matrix Subungual hyperkeratosis with heaped up thickened scale material may be present or the nail plate may be lifted from its bed by the moist macerated skin

The fingernails and toenails may be involved concurrently or separately Usually more than one nail is involved but it is rare to see affection of all ten nails of the hands or feet Complete examination often reveals involvement of the glabrous skin as well as of the nails especially the interdigital spaces of the toes

Diagnosis—Psoriasis pyogenic and monilial infections must be differentiated In psoriasis (p 3414) there is more frequently involvement of all the nails and coincidental typical lesions may be present on the glabrous skin There are two ordinary forms of psoriasis of the nails one in which the nail plate is somewhat irregular and is profusely pitted resembling somewhat the side of a thumb (thumbing of the nail) In the second there are actual subungual psoriatic papules which begin near the cuticle and work towards the free border The nail itself may be unaffected or may show some irregularity and discoloration Pyogenic organisms attack the soft tissues about the nail and produce a paronychia (p 3973) Secondary nail involvement may occur with some distortion and grooving as a result In monilial disease a practically constant accompaniment to the deformity of the nail is the chronic paronychia Chronic paronychia may occur with onychomycosis but less regularly than with monilial infections

Treatment—See p 3307

RINGWORM OF THE EXTERNAL AUDITORY CANAL (OTOMYCOSIS MYRINGOMYCOSIS)

Otomycosis is evidenced by redness swelling and a serous discharge from the auditory canal At times the disease is mild and dry with redness and scaling alone Involvement may include the entire external auditory canal the ear drum and the outer surface of the pinna

There is some doubt that this condition is truly a primary fungous infection Various types of organism can be found especially *Aspergillus* and *Monilia* At times streptococci are present Some investigators believe that these are secondary invaders The disease is seen in adults chiefly and in either sex

Seborrheic dermatitis and contact dermatitis must be differentiated Seborrheic dermatitis (p 3432) usually involves the postauricular surfaces and the scalp as well as the ears Contact dermatitis (p 3330) must be eliminated by exclusion of all possible excitants

Treatment—See p 3307

RINGWORM OF THE AXILLARY HAIR (LEPOTHRIX TRICHOMYCOSIS AXILLARIS)

Ringworm of the axillary hair is a fairly common condition which is asymptomatic but constitutes a blemish noticed by the patient The axil

RINGWORM OF THE BEARDED REGION (TINEA BARBAE TINEA SYCOSIS BARBER'S ITCH)

Barber's itch is rarely encountered in this country. It is essentially a *fungous infection* of the *hair follicles* of the *beard*. The usual variety consists of one or more tender reddened infiltrated or softish circumscribed swellings which resemble pyogenic infections. They most commonly involve the chin or sides of the face and simulate closely kerion of the scalp. This type is caused by *M. lanosum* or *Trichophyton gypseum* and is acquired from diseased animals. The *trichophyton reaction* (p 3299) is usually strongly positive. The less frequent variety is caused by *Trichophyton violaceum* or *Trichophyton purpureum* and produces a condition resembling pustular folliculitis. The *trichophyton reaction* is usually negative in the latter.

Diagnosis—In differential diagnosis ordinary pyogenic infections *syccosis barbae*, *iododerma* and *bromoderma* are considered. *Furuncles* and *carbuncles* (p 3248) tend to be more acute, more painful and tender and form pus. *Sycosis barbae* (p 3249) frequently attacks the upper lip and unlike in tinea infections it is difficult to pluck the hairs. In the eruption due to the *halogens* there is usually a definite history of ingestion of the drug and lesions are likely to be found on other portions of the body. In doubtful cases *examinations for the fungi* are of importance.

Treatment—See p 3307

FAVUS

Favus is a special type of ringworm of the scalp rarely seen in the United States but very frequently observed on the continent of Europe. The causative fungus is the *Achorion schoenleini* which can be found fairly readily in direct examination and grown in artificial culture media.

Clinical Appearance—The ordinary variety is very characteristic showing numerous *cup shaped sulfur-yellow dry crusts on the scalp*. These crusts (*scutula*) are concave on their outer surface while the side towards the scalp is convex and pressed firmly into the scalp. It is believed that this pressure of the crust produces local atrophy with destruction of the *hair follicles* so that there remain permanent *alopecic scarred areas* after the disease is cured.

In another form there is marked scaliness, the scales being rather firmly attached but crusts are absent. This type is easily mistaken for seborrheic dermatitis. Unlike the crusted form atrophy and baldness do not occur.

The crusted variety of favus is seen mostly in children although it may extend into adult life. There is not the same tendency to spontaneous cure at puberty as observed in tinea capitis. The scalp seborrheal form is seen more commonly in adults. The disease may also attack the glabrous skin and the nails.

Treatment—See p 3307

RINGWORM OF THE NAILS (ONYCHOMYCOSIS)

The toenails and the fingernails may be infected by various fungi. Changes produced by *Monilia albicans* (p 3301) and by *Achorion schoenleini* (p 3304) have already been briefly discussed. The present discussion

TINEA IMBRICATA

Tinea imbricata is a chronic superficial fungous infection of the skin which is endemic in tropical countries. It is encountered commonly in the South Pacific islands and in India. The parasite is a vegetable fungus of the *Endodermophyton* family and the disease is transmissible by direct bodily contact.

In its characteristic state the eruption consists of concentric rings formed of partially freed scales which overlap one another whence the term "imbricated." The lesions have a brownish color. The process begins as a single point of infection which develops into a ring from whose border another ring develops. The repeated occurrence of this phenomenon leads to the fully developed picture of numerous concentric rings. The trunk and extremities are principally affected but involvement of other areas including the nails has been reported. The condition is chronic and recurrence is common.

Treatment—See p. 3307

Treatment of Superficial Dermatophytoses and Dermatomycoes

The treatment of superficial fungous infection requires preparation for the use of the fungicide, application of the specific and measures to prevent recurrence.

Preparatory Treatment—Before application of the fungicide the infected area must be cleansed, shaved and cleared of the complicating treatment dermatitis. For purposes of cleansing nothing can compare with vigorous scrubbing with soap and water. Hairy areas are dry shaved with an electric razor. For cosmetic and psychologic reasons it may not be advisable to shave the hair of the head but it should be close cropped or bobbed. Individually infected hairs are manually epilated with a forceps. The vibrissae of the external auditory canal in an otomycosis are most satisfactorily destroyed by local application of radium. The problem of dealing with onychomycosis is made difficult by the inaccessibility of the invading organism. The fungicide can have little effect unless the nail is completely scraped on its surface and around and beneath the cuticle.

Until a treatment dermatitis has been dissipated there is little hope for successful fungicidal activity. It is a prime consideration for the practitioner to get rid of supplementary problems before attacking the underlying cause. In the presence of an acute dermatitis wet dressings are applied of sterile saline, cold boiled milk or a solution of penicillin improvised by dissolving the contents of a vial containing 100,000 units in 1,000 cc (one quart) of boiled water. Between moist applications the involved area is protected by White Petroleum Jelly or an ointment of penicillin made so that each gram contains 1000 to 3300 units.

Fungicidal Therapy—The fungicides of choice should be those preparations which possess greatest activity against the pathogen and least threat to the tissues of the host. In order our preferences include

- 1 Sodium and Calcium Propionate (Sopronol)
- 2 Undecylenic Acid (Desenex)
- 3 Cresatin
- 4 Compound Ointment of Benzoic Acid (Whitfield)

DIFFERENTIAL DIAGNOSIS OF

Dermatoses of the Ear

The commoner dermatoses of the ear include frostbite postauricular sebaceous cysts and the painful furunculosis of the external auditory canal

CLINICAL MANIFESTATIONS AND
DIAGNOSTIC FEATURES

| | |
|---|---|
| Chilblain | Frostbite producing areas of cyanotic edema associated with severe itching and pain |
| Comedones | Blackheads usually situated within outer portion of external auditory canal may predispose to furunculosis |
| Contact Dermatitis | Usually lobular due to earrings Postauricular from spectacles or hair dye (p 3162) |
| Epithelioma | Isolated persistent papule with tendency to growth and ulceration More frequent in males beyond the age of 40 (p 3220) Get biopsy |
| Erysipelas | Characteristic fiery red lesion with indurated advancing border moderate to severe pain and systemic manifestations (p 167) |
| Furunculosis of External Auditory Canal | Severely painful pyoderm with marked edema which may obstruct external auditory canal and spread over mastoid region and temporal bone (p 2112) |
| Herpes Zoster | Vesicular eruption producing severe pain (p 435) |
| Impetigo Contagiosa | Staphylococcal pyoderm with crusting Usually seen in infants and young children (p 3251) |
| Intertigo | Usually postauricular due to pressure of spectacles or to associated seborrhea (p 3161) |
| Keloid | Usually lobular following puncture for ear rings (p 3166) |
| Lupus Erythematosus | Papular eruption with characteristic adherent scales containing projections on under surface Healing with atrophy Associated with cardiovascular renal manifestations (p 3399) Get biopsy |
| Lupus Vulgaris | Persistent apple jelly nodules Ulceration rare Healing with scar formation (p 3262) Get biopsy |
| Otomycosis | Eczema of external auditory canal with local itching discharge and pain (p 2143) Identify fungus in smears |
| Sebaceous Cysts | Painless tumor usually found in lobular or postauricular region Puncture yields caseous material (p 3208) |

lary hairs are studded with very firm adherent nodules of yellow red or black color A species of *actinomyces* has been demonstrated in the lesions

The absence of pediculi and pruritus and the color of the lesions readily differentiates this condition from *pediculosis pubis* which has spread to the axilla

Treatment—See p 3307

- 3 Put the shoes in a paper box close the lid and wrap in paper
- 4 At the end of at least six hours remove the shoes from the box and air for twenty four hours
- 5 Apply saddle soap inside and outside of all parts of the shoe
When the soap is dry polish as usual

The feet are best given *prophylactic treatment* morning and night The value of foot baths has been greatly overemphasized The following routine is suggested

- 1 Scrub the feet with soap and water Dry between and under the toes
- 2 Powder with talc or preferably 15 per cent calcium propionate (*Sopronol*)
- 3 Wipe off the excess of powder
- 4 If the feet tend to sweat excessively separate the toes with lamb's wool or wisps of absorbent cotton

Desensitization—Desensitization with Trichophytin has been attempted particularly in the treatment of the dermatophytids Our results have not been encouraging but immunization therapy may be utilized as a measure of desperation

THE DEEP DERMATOPHYTOSES AND DERMATOMYCOSES

The deep fungus infections are essentially systemic diseases However the *portal of entry* may present a *characteristic dermatous and localizing cutaneous eruptions* often signalize the clinical condition The broader and more general features of the deep fungus infections are elsewhere considered (p 485) In this section only the cutaneous manifestations are described

ACTINOMYCOSIS

In actinomycosis the skin is rarely primarily affected though commonly and characteristically it shows secondary lesions In the *primary cutaneous form* there appears an indurated tender red swelling which breaks down discharges purulent material and eventuates in a progressively enlarging and deepening ulceration

The *secondary cutaneous form* is most often seen and most readily recognized as a sequence of infection of the *jawbone* which presents a diffuse deep red brawny swelling over the mandible and which arises from a primary infection of the bony structure This curious site for the lesion may result from the habit of chewing on straw or grain contaminated with the ray fungus In other instances the skin is secondarily involved from an infected abdominal or thoracic viscus

Actinomycosis of the mandible is chronic and indolent and as a rule not associated with important systemic symptoms Nodular enlargements appear in the edematous areas These break down and form sinuses which discharge purulent matter Examination of the pus discloses the presence of the characteristic tiny *sulfur granules* These are small yellowish oatlike structures found to be composed of masses of fungi

Treatment—See p 3316

- 5 Castellani Paint (p 3126)
- 6 Liquor Carbonis Detergens (3 to 20 per cent) in a Paste of Zinc Oxide
- 7 Ointment of Ammoniated Mercury (2 to 5 per cent)
- 8 Resorcinol (3 per cent) in Calamine Lotion
- 9 Salicylic Acid (3 per cent) and Oil of Cade (3 per cent) in equal parts of Lanolin and Petroleum Jelly
- 10 For small isolated patches 3.5 per cent Tincture of Iodine 1 per cent Gentian Violet (especially in moniliasis) or 1 per cent Thymol
- 11 Tincture of Benzoin containing 25 per cent Resorcinol or 5 to 10 per cent Chrysarobin ointment in Tinea Imbricata

It may be stated regretfully that DDT penicillin and sulfonamides possess negligible fungicidal activity

Our custom, after preliminary treatment has been completed is to suggest the use of Sopronol in powder and ointment form as marketed. The ointment is used at bedtime removed in the morning and replaced by the powder. The powder is dusted off so that it does not cake. Toes or other opposing surfaces are separated by wisps of lamb's wool.

Treatment failures are exposed to one of the other fungicides particularly Desenex Cresatin or half strength Whitfield's ointment. Should the mycosis still prove resistant, the patient is urged to remain at home for intensive therapy with Castellani Paint or Resorcinol (3 per cent) in Calamine Lotion. For a moniliasis 10 per cent ointment of sodium caprylate is suggested.

Finally with obstinate persistence or recurrence the patient is referred to the specialist for roentgen therapy. Such an eventuality is the expectation in onychomycosis otomycosis favus Trichophyton purpureum infections and tinea capitis.

Prophylaxis—Successful fungicidal therapy must be followed by preventive treatment. Indeed reinfection and auto inoculation must be stopped concurrently if success is to be achieved. The measures to be employed include anidrosis to deprive fungi of necessary cultural conditions and sterilization of wearing apparel particularly socks and shoes.

To achieve dryness of body surfaces many devices may be required. The obese must lose weight opposing surfaces (thighs breasts or toes) must be kept powdered and separated by lamb's wool hyperidrosis of hands and feet may be lessened by frequent soaks and application of hand lotion (p 3142) soiling of the perineum may be diminished by measures of local hygiene and corrections of conditions leading to incontinence of urine and feces (p 2265) maceration of the hands may be prevented by the use of gloves while dishwashing etc maceration of the feet may be lessened by frequent changes of socks and the use of corrective shoes (p 3081).

Sterilization of footwear must be undertaken in the treatment of athlete's foot. For the household sterilization of a few pairs of shoes the following method is suggested.

- 1 Cleanse shoes with soap and water and dry with a cloth
- 2 Place a pad of cotton in shoes and spread it out to make an insole. Pour on one teaspoonful of formaldehyde U.S.P. dilute 1 to 10 with water.

and have the odd gyrate or geometric pattern the serologic test of the blood is usually positive and the lesions respond promptly to antisyphilitic treatment. Ultimately the diagnosis of blastomycosis must be made by demonstration of the presence of the fungus by direct examination by cultural or histopathologic studies.

Treatment—See p 3316

SPOROTRICHOSIS

Sporotrichosis is an unusual fungous disease caused by the *Sporotrichum schenckii* less often by other sporotricha which are found in vegetable matter and in human and animal waste. Perhaps the most important



Fig 966—North American blastomycosis of leg showing an open granulating ulcer*

manner of infection is by inoculation of the parasite into minor cutaneous wounds from infected vegetation.

Clinically the primary sore resembles the syphilitic chancre (p 3278). However there is usually pus formation and subsequently an indolent ulcer develops. Most important there soon appears an indolent lymphangitis extending from the primary sore. Nodules form in the course of the inflamed lymphatics. These nodules break down and assume the characters of the primary sores. The regional lymph nodes are rarely affected. The organism is difficult to demonstrate by direct examination of material from lesions but it is readily grown on artificial culture media (p 488).

BLASTOMYCOSIS

The blastomyces attack the skin primarily and secondarily. The *localized cutaneous form* originates as a *papule* which soon becomes pustular and crusted. As extension occurs by direct spread to the surrounding skin the lesion assumes a rather typical appearance. It consists of a raised flat-tended encrusted *disl* whose border is studded with a great number of minute *abscesses*. Puncture of one of these lesions releases a small amount of fluid pus in which the organism can most readily be found. Beneath the crust is a small amount of thin, purulent material which bathes a surface which is distinctly verrucous in character. Healing first occurs in the older



Fig. 96 —Actinomycosis of the face. Note swelling of subcutaneous tissues and multiple sinus formation.*

areas so that a central scarred area is found surrounded by an active advancing ring shaped border (Fig 966)

Under the microscope the blastomyces are recognized as round *double contoured cells* with characteristic *budding*. The buds are new organisms reproduced from the parent cells and ready for separation.

Blastomycosis must be differentiated from tuberculosis, *verrucosa cutis lupus vulgaris* and the nodulo ulcerative syphiloderm. *Tuberculosis verrucosa cutis* (p 3259) is dry, not crusted, more indurated and lacks the minute abscesses in the border. It is most often observed on the dorsum of the hand. *Lupus vulgaris* (p 3262) is most common on the face, is dry, contains the diagnostic apple jelly nodules and also lacks evidence of abscesses. *Late syphiloderms* (p 3263) are usually dry, never show abscesses.

Tuberculosis of the skin (p 3258) does not produce chainlike lymphatic swelling. Cultures are negative and the biopsy reveals the distinctive histopathology. In *tularemia* (p 323) regional adenopathy is constant; there are systemic symptoms; the skin test (Foshay) and serum reactions are positive.

Treatment—See p 3316



Fig 968—Progressive coccidioidomycosis. Note large subcutaneous abscess and draining sinus.

COCCIDIOIDAL GRANULOMA

Coccidioidal infection is most often seen as bronchopneumonia or influenza-like episodes known as Desert Fever, Valley Fever, or San Joaquin Valley Fever (p 499). The cutaneous manifestations of this infection are of minor importance. During the bronchopneumonic infection a typical *erythema nodosum* eruption usually appears one to two weeks after the onset (Fig 968).

Microscopic study of the cultures reveals the organism as threadlike mycelia with lateral branches tipped with clusters of spores

Diagnosis—Syphilis tuberculosis tularemia and pyogenic infections may be confused with *sporotrichosis* The *chancre of syphilis* (p 3278) is



Fig 967—Primary sporotrichosis of the thumb *

not productive of free pus The regional lymph nodes are involved early and darkfield studies reveal the treponema The *gumma of syphilis* (p 3286) does not involve the lymphatics there is no satellite adenopathy, cultural investigations are negative the serologic tests of the blood are positive and the response of the gumma to specific treatment is prompt

* Conant and Smith Manual of Clinical Mycology

Differential diagnosis from other granulomas requires cultural and tissue studies

Treatment—See p. 3316

TORULOSIS

Torulosis is a rare disease which most commonly localizes in the central nervous system and which rarely affects the skin. The cutaneous manifestation is an abscess like *granuloma* which has no particular morphological characteristics and is only recognized when the etiologic agent, the *Torula histolytica*, an ubiquitous yeast, is demonstrated in cultures direct

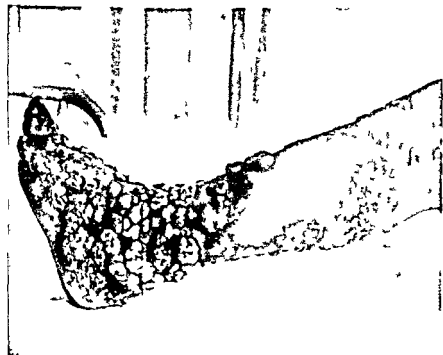


Fig. 970—Chytridiomycosis of the leg caused by *Hormodendrum pedoso*. Note the cauliflower-like lesions and the edema of the leg.

examination of pus or in the sections of diseased tissue. The organism appears as a rounded encapsulated cell with budding suggestive of blastomycetes.

MADUROMYCOSIS (MYCETOMA)

The mycetoma is an infection caused by actinomycetes. It is very common in India but rare in North America. It usually follows infection of some minor wound of the foot (*Madura foot*). There occurs swelling of the part which becomes dull red in color and covered with *nodules* penetrated by multiple discharging *sinuses*. The discharge is thick, foul smelling, viscous and contains *granules* of various colors. (Fig. 969)

Treatment—See p. 3316

After Carrion and Koppisch. Puerto Rico Journal of Public Health and Tropical Medicine 9

Infection of the skin by the *Coccidioides immitis* is characterized by indolent abscesses which rupture and produce chronic ulcers or gumma like masses. In these the fungus is easily demonstrated as a double contoured round body larger than the blastomyces. There are no lateral buds such as characterize the latter but within the body a variable number of small round endospores are demonstrable. These are freed when the organism ruptures and then they go on to develop into mature parasites. The fungus is readily grown on artificial culture media (p 488) from which the antigen coccidioidin has been prepared. When inoculated intradermally into persons affected with this disease an inflammatory papule is produced.



Fig 969—Maduromycosis of the foot caused by *Monosporium apiocephalum*. Note the swelling of the foot and the multiple discharging sinuses.

This indicates an allergic state which is specific since inoculation of uninfected persons invariably produces no reaction.

Treatment—See p 3316

PARACOCCHIDIOIDAL GRANULOMA (ALMEIDA'S DISEASE)

Almeida's disease is seen exclusively in South America and is caused by the *Paracoccidioides brasiliensis*, an organism closely related to but different from the *Coccidioides immitis*.

The infection occurs about the mouth where it produces an indolent inflammatory swelling which often but not invariably ulcerates. Well marked regional lymphadenopathy is present.

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four divided portions Sulfonamide failure; finally may be treated with antimony as described in the next paragraphs dealing with leishmaniasis and filariasis

CUTANEOUS LEISHMANIASIS AND HELMINTHIASIS

Skin and subcutaneous tissues may be invaded by parasites such as the *Leishmania* and by round worms of the filarial variety. In some instances the lesion appears to be local as in American leishmaniasis but other disturbances such as filariasis are definitely systemic with secondary cutaneous phenomena

AMERICAN LEISHMANIASIS (ESPUNDIA)

Espundia is a chronic ulcerative disease of the skin and mucous membranes which is endemic in the western hemisphere and is caused by a



Fig 271—American leishmaniasis (Case observed in Sao Paulo, Brazil by Dr. Howard Fox)

specific *leishmanial* parasite. The disease is encountered chiefly in the southern portion of Mexico, in Central America and in the northern half of the South American continent. The causative agent is the *Leishmania brasiliensis*. Although the exact mode of transmission is unknown, sand flies of the *Phlebotomus* family are strongly suspected of being intermediate hosts. Contact infections are also believed to occur.

Cutaneous Manifestations—The exposed surfaces of the body are usually affected most commonly the face and other parts of the head but in instances of involvement of the extremities are recorded. The *primary lesion* is an infiltrated erythematous area which breaks down and forms an *indolent ulcer*. The ulcer has a raised purplish edge and is covered with a crust beneath which there oozes a thin purulent material. The lesion may heal in a period of a few months to several years and produce a *disfiguring scar*. While the cutaneous disturbance is still active or after it has

CHROMOBLASTOMYCOSIS

Rare but increasing numbers of instances of chromoblastomycosis are being reported in the United States the West Indies and South America. The causative agent is a fungus *Hormodendrum pedrosoi* or others which probably gains access after trauma.

The leg is almost always affected. The infection is indolent leading to hypertrophic verrucous masses resembling mushrooms or small cauliflower flowers. Ulceration may ensue with foul purulent discharge. The color of these lesions is variable pink dull red purplish or brown (Fig. 970).

The condition must be differentiated from *tuberculosis cutis verrucosa* (p. 3259) and *blastomycosis* (p. 3310) primarily. Usually laboratory examinations are necessary to make a final diagnosis.

Treatment—See p. 3316

Treatment of Deep Dermatophytoses and Dermatomycoses

Local treatment of deep fungous infections is unsatisfactory even when pursued competently persistently and conscientiously. The practitioner errs if he fails to include intensive systemic therapy in his routine since widespread dissemination must occur with greater frequency than is generally suspected.

Local Treatment—Before the nature of the infection is established the local treatment suggested for superficial fungous lesions merits trial (p. 3307). With persistence of the granuloma various measures are instituted for the purpose of local destruction of involved tissue. These include local excision curettage under anesthesia freezing with dry ice electrocoagulation electrodesiccation and roentgen therapy. Amputation may be necessitated as a measure of desperation in maduromycosis.

Following tissue destruction the Russians report splendid results after applications of *aloe vera* as elsewhere described (p. 3118).

Systemic Treatment—Systemic therapy must be considered in all but the most trivial deep fungous infections. In the eras that preceded antibiotic treatment main reliance was placed on the iodides. Initial doses of 2 to 5 drops of saturated potassium iodide were increased by increments of 3 drops daily until the patient received as much as 5 gm. (300 grains) each day provided that incapacitating manifestations of iodism were not encountered.

Iodide therapy is of greatest value in sporotrichosis where complete cure may be anticipated. It is of almost equal value in some instances of blastomycosis and may be useful in actinomycosis.

Following iodide therapy or in conjunction with it penicillin should be given intensively using intramuscular injections of 50,000 to 100,000 units every three hours. In actinomycosis penicillin has specific effects in other fungous infections it eliminates secondary infection by sensitive organisms.

Treatment failures are entitled next to a course of sulfonamide. To achieve a high initial concentration the patient may be given an oral dose of 5 gm. of sulfadiazine and an intravenous injection of 100 cc. of 5 per cent sodium sulfadiazine. Thereafter following the usual precautions (p. 88) maintenance daily doses of 6 to 8 gm. are ordered by using

(Neostam) is given intravenously in a freshly prepared 2 per cent aqueous solution. The initial dose is 2.5 cc each succeeding dose being increased by 2.5 cc up to the maximal dose of 10 cc. A total of twelve to fifteen doses is administered. *Potassium antimony tartrate* freshly prepared in 2 per cent aqueous solution may also be given. The initial dose is 2 cc each succeeding dose being increased by 1 cc up to the maximum dose of 5 cc. A total of twenty to forty doses of this drug may be administered. Whichever drug is used the injections may be made two or three times weekly provided that no toxic reactions occur. Local treatment employs the topical application of dry ice or the injection into the edges of 2 cc of 1 per cent berberine sulfate.

TROPICAL CUTANEOUS LEISHMANIASIS (ORIENTAL SORE, ALEPPO BOIL, DELHI BOIL)

Cutaneous leishmaniasis is a specific infectious disease endemic in the Mediterranean basin. It is caused by the flagellate *Leishmania tropica*.



Fig. 93.—*Leishmania tropica*—Aleppo boil. (Courtesy of Dr. J. Ra. Jah. Beirut, Syria.)

(p. 534). The disease is common among the natives living in poor hygienic surroundings in the countries of the Levant and northern Africa. It is transmitted to humans by the bite of a *sandfly* (*Phlebotomus*) and is probably most often transferred from man to man. Dogs and cats are also believed to harbor the infection.

Clinical Manifestations.—The condition is most common on the face where it begins as a red *papule*. This becomes crusted and later ulcerates. The ulcer is sharply margined, shallow, saucer-like and indolent, healing in time and leaving a thin atrophic scar. Occasionally a disfiguring scar remains. One attack apparently immunizes against subsequent reinfection. The natural tendency of the condition is towards spontaneous cure in from six to eighteen months (Fig. 972).

Diagnosis and Treatment.—While the individual lesions might well be mistaken for syphilis, tuberculosis or pyoderma, the frequency of the eruption in endemic areas suggests the diagnosis, which is proved by the demonstration of the *Leishman Donovan* bodies (Fig. 972, p. 3318).

completely healed ulcers may develop in the nose or pharynx. These commence as indurated areas which necrose and eventuate in chronic granular ulcers. Regional lymphadenopathy is generally present. Death may result from interference with respiration secondary pyogenic infection or pulmonary infection (Fig 971)

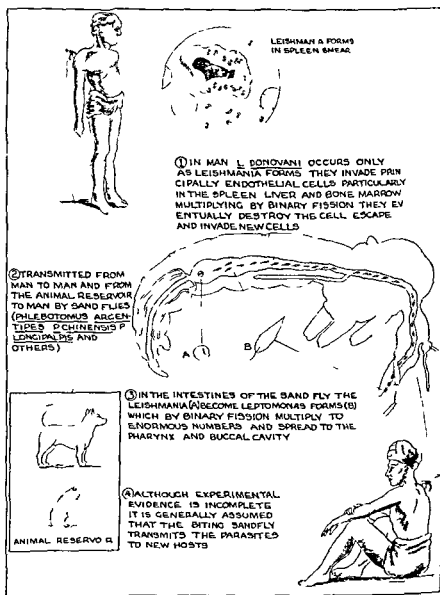


Fig 972.—Life cycle of *Leishmania donovani*.

Diagnosis—The diagnosis is readily made by demonstration of the parasites in the ulcers. They are found most easily in the early lesions underneath the edge of the ulcer and can be seen in smear or biopsy.

Treatment—The treatment of espundia is similar to that of oriental sore (cutaneous leishmaniasis) and kala azar (p 3320). *Stibamine glucoside*

of treatment is repeated only if cure is not obtained and after interval of one month. Some patients show fall of blood pressure, syncope and fainting after first or second injection. This may be prevented or relieved by injection of a small dose of epinephrin. The administration of calcium and glucose during treatment may protect the liver from damage.

FILARIASIS

Helminthic diseases (p. 537) of the subcutaneous tissues are produced by certain of the roundworms and the guinea worms *Dracunculus medinensis*. The roundworms invade in the microfilarial state. These immature or prelarval forms are produced by the adult females of *Wuchereria*, *Loa*, *Onchocerca*, *Acanthoecilonema* and *Mansonella*.

Life Cycle.—The tissue nematodes of man have an arthropod intermediate host. With the filarial worms the intermediate hosts are diptera or flies while *Dracunculus* uses cyclop, a crustacean.

The adults of all forms live in the tissues of man. Instead of laying eggs they produce the microfilariae. The microfilariae may make their way to the peripheral blood to be picked up in the blood meal of the intermediate host. They may migrate to the surrounding tissue to be ingested by the blood sucking intermediate host or they may be deposited in small ulcers which burst on contact with water.

Within the intermediate host the microfilaria sheds its sheath and penetrates the wall of the gut. It is carried to the thoracic muscles where it undergoes molting before coming infective. It then migrates to the mouth parts of the insect and escapes from the proboscis when the skin of man is bitten. The injected larva penetrates the subcutaneous tissue, matures and mates with a member of the opposite sex. The female produces microfilariae which pass by way of the lymphatics to the lymph glands where they develop to adult form. Other mature in the subcutaneous tissues.

Clinical Manifestations.—The better known clinical manifestations of invasions by the tissue roundworm include *filariasis loiasis onchocerciasis* and *dracontiasis*.

Filariosis (Filarial Elephantiasis).—Filariosis is caused by *Wuchereria bancrofti*. It has worldwide distribution in tropical and subtropical countries. A focus of infection existed in Charleston, South Carolina.

The adult filaria live in the lymphatic vessels and glands. Here they give birth to large numbers of sheathed microfilariae which migrate into the blood stream where they live as long as two weeks. When the microfilariae are taken up in the blood meal of the intermediate mosquito host the life cycle continues. The microfilariae leave their sheaths in the stomach and develop into infective forms which elongate to wormlike larval filariae. The latter migrate to the head and proboscis of the mosquito where they emerge onto the skin of the man who is bitten by the insect. They then return to the lymphatics and lymph glands where they develop to the adult stage and complete their cycle. The most important vectors are *Culex fatigans*, a cosmopolitan night biting mosquito and *Aedes taeniorhynchus*, a day biting mosquito found on the islands of the Pacific.

In the regions where the disease is epidemic the diagnosis of filariasis may be suspected before actual demonstration of the organism is possible. The premonitory manifestations include anorexia, nausea and vomiting and pains in the groin, testicles and inner aspects of the thigh. At this time laboratory examinations may show a pronounced eosinophilia but microfilariae are not demonstrable in the circulating blood.

Treatment with antimony (p 132) is effective. It is administered as in American Leishmaniasis (p 3319). Recently rapid cure of the lesion has been reported after daily application of *sulfapyridine powder* directly to the ulcer.

In the Egyptian Sudan and some adjacent areas leishmaniasis is resistant to treatment with antimony preparations. In these regions and elsewhere if the response to neostibosan is poor stilbanudine isethionate

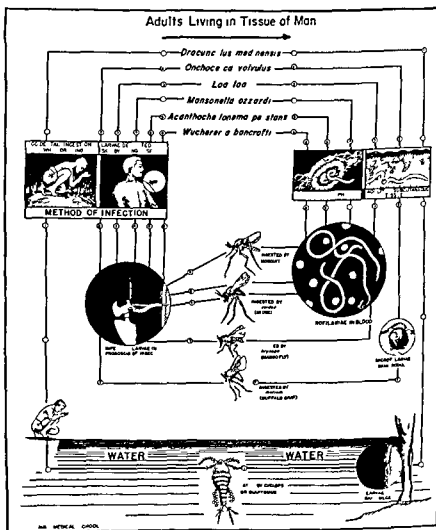


Fig 974—Life cycles of important human roundworms

(44 diamidino stilbene isethionate) is used intravenously. The drug must be used in a *freshly prepared* solution in 10 cc of sterile distilled water without heating. The water must be neutral or very slightly acid (pH 6.8-7.2). Old solutions may cause severe late toxic effects on the liver, kidneys or pancreas even after completion of a course of treatment. The dose is 10 milligram (0.001 gram) per kilogram of body weight (maximum adult dose 0.15 gram) every other day for 15 injections. The course

of treatment is repeated only if cure is not obtained and *after interval of one month*. Some patients show fall of blood pressure syncope and fainting after first or second injection. This may be prevented or relieved by injection of a small dose of epinephrin. The administration of calcium and glucose during treatment may protect the liver from damage.

FILARIASIS

Helminthic diseases (p. 537) of the subcutaneous tissues are produced by certain of the roundworms and the guinea worms *Dracunculus medinensis*. The roundworms invade in the *microfilarial state*. These immature or prelarval forms are produced by the adult females of *Wuchereria*, *Loa*, *Onchocerca*, *Acanthoecilonema* and *Mansonella*.

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The adults of all forms live in the tissues of man. Instead of laying eggs they produce live *microfilariae*. The *microfilariae* may make their way to the peripheral blood to be picked up in the blood meal of the intermediate host. They may migrate to the surrounding tissues to be ingested by the blood-sucking intermediate host or they may be deposited in small ulcers which burst on contact with water.

Within the intermediate host the *microfilaria* sheds its sheath and penetrates the wall of the gut. It is carried to the thoracic muscles where it undergoes molting before coming infective. It then migrates to the mouth parts of the insect and escapes from the proboscis when the skin of man is bitten. The injected larva penetrates the subcutaneous tissue, matures and mates with a member of the opposite sex. The female produces *microfilariae* which pass by way of the lymphatics to the lymph glands where they develop to adult form. Others mature in the subcutaneous tissues.

Clinical Manifestations —The better known clinical manifestations of infections by the tissue roundworm include *filariasis*, *loiasis*, *onchocerciasis* and *dracontiasis*.

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Ten to fourteen days after the premonitory symptoms the more characteristic manifestations appear of acute epididymitis funiculitis varico-



Fig 975—Filariasis thickened spermatic cord *



Fig 976—Filariasis varicose inguinal lymph nodes †

cele swelling of the testicle and scrotal swelling. Later the patient develops involvements of superficial lymph nodes and vessels in the groin axilla and epitrochlear regions.

Mackie: Manual of Tropical Medicine

† After Taniguchi—Kumamoto in Strong: Still's Diagnosis, Prevention and Treatment of Tropical Diseases. The Blakiston Company

In association with organic manifestations most American soldiers infected with the filaria in the recent Pacific campaign developed pronounced anxiety symptoms since there was general belief that the disturbance resulted in impotence and sterility. Fortunately neither of these sequels need be anticipated.

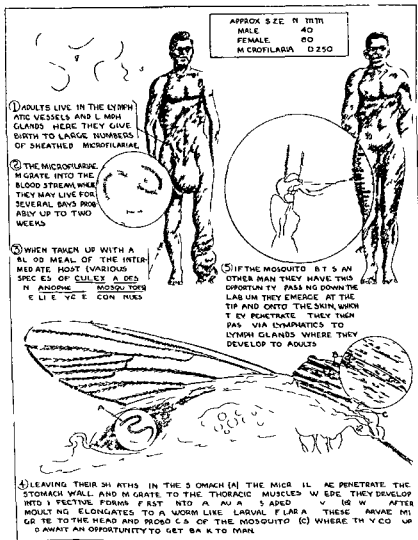


Fig 9-7—Life cycle of Wuchereria bancrofti

In neglected late infections there develops the characteristic picture of elephantiasis of scrotum, vulva and legs. Less frequently arms and breasts are involved. Many patients develop chyluria, chylous ascites or chylous diarrhea. Episodes of elephantoid fever occur at irregular intervals of

Larval form isolated from Faint Human Histopathology U.S. Naval Medical School by C. A. B. Ke

weeks months or years When the fever is announced by a sudden chill there is a suggestion of the diagnosis of malaria

Diagnosis—The positive diagnosis in filariasis is made by identification of microfilaria in blood smears taken between the hours of 10 P.M. and 2 A.M. A concentration technic has been devised by which 1 cc of blood

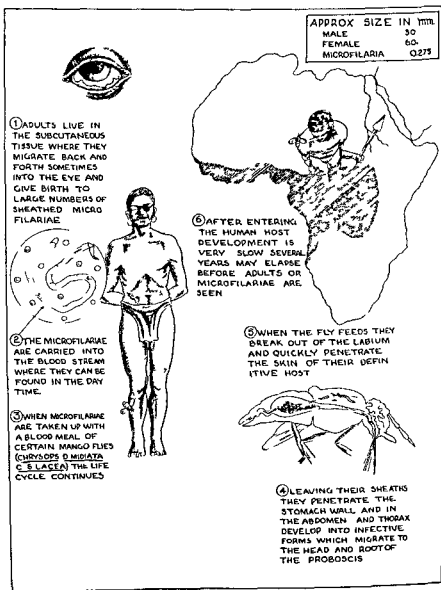


Fig 978—Life cycle of *Loa loa* *

is taken from the vein and discharged into 10 cc of 2 per cent formalin. The mixture is placed in a conical tipped centrifuge tube mixed thoroughly and permitted to sediment for twelve to twenty four hours. At the end of this time the supernatant fluid is decanted the sediment is

* Adult in bulbar conjunctiva modified from Faust Human Helminthology Naval Medical School by C A Baker

smear on a glass slide and stained with Loeffler's methylene blue for two or three minutes rinsed and then counterstained for one or two minutes with $\frac{1}{2}$ per cent aqueous eosin or Giemsa. The parasite also may be demonstrated by similar technic from hydrocele fluid, ascitic fluid or biopsy specimens. A skin test with an antigen of $\frac{1}{4000}$ *Dirofilaria immitis* and a complement fixation reaction also are available.

Treatment—Main reliance in the treatment of filariasis is placed on antimony preparations. Experiences during the recent war seem to indicate that Lithium antimony thiomalate (Anthiomaline) holds greatest promise. The drug is supplied as a 6 per cent solution. Injections are given intramuscularly in doses of 3 cc, although the initial injection is preferably only 1.5 cc in order to test for sensitivity. Treatment is continued daily for approximately a month, the efficacy of therapy being estimated by making microfilaria counts on 0.1 cc of blood. A sharp reduction in the microfilaria count is noted within the first week, however if treatment is discontinued prematurely the infection recurs. Injections of Anthiomaline may produce toxic manifestations. Vomiting occurs in 40 per cent, an occasional febrile reaction is encountered, toxicoderms are not infrequent.



Fig 979—Sheathed microfilaria of *Loa loa* in thick blood film. Caudal nuclei extend to tip of tail. (From slide of Liverpool School of Tropical Medicine loaned by Puerto Rico School of Tropical Medicine.)

local reactions are noted at the site of injection and an occasional patient complains of arthritic pain. Transitory and slight anemia or an albuminuria may be reported by the laboratory.

LOIASIS (CALABAR SWELLING)

Loiasis is a condition of tropical West Africa, particularly seen in the Congo River country. The condition is caused by the *Loa loa*, a filaria which lives in the subcutaneous tissues. Their microfilariae find their way into the blood stream with diurnal periodicity. The intermediate host is a fly of the genus *Chrysops*.

The adult *Loa loa* lives in the subcutaneous tissues where it migrates back and forth, sometimes into the eye and gives birth to a large number of sheathed microfilariae. These are carried into the blood stream where they can be demonstrated during the daylight hours. When the microfilariae are taken up with the blood meal of the flies, the life cycle continues. The infective forms develop and migrate to the head and root joints. When the flies feed they quickly penetrate the skin of

their definitive host. Here they develop slowly over the course of several years and produce migratory *subcutaneous swellings* (calabar swellings) which are characteristically seen under the conjunctivae.

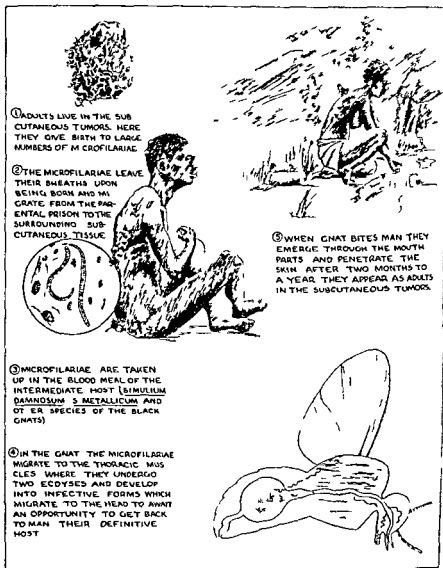


Fig 980—Life cycle of *onchocerca volvulus*

The diagnosis of loiasis is made by the demonstration of microfilariae in the blood. Occasionally the adult may be extracted from under the skin or the conjunctivae and identified.

Treatment—See p 3325

ONCHOCERCIASIS (THE BLINDING FILARIA)

Onchocerciasis is a chronic parasitic disease which is endemic in certain tropical countries and characterized by the formation of *subcutaneous*

* A fullis in tumor after Brumpt from Cl in Her. In tract on to Parasitology U S Natl Medical School by C A Baker

ous nodules The causative organism is the *Onchocerca volvulus* and the insect vector is a black gnat (*Simulium*). The gnat ingests the embryos by biting an infected human being. After a short developmental period the insect is capable of transmitting the infection to others. The microfilariae are introduced into the insect bite and form a subcutaneous tumor in which they develop into adult worms. From these tumors microfilariae are released and spread into the lymphatic vessels and adjacent tissues but only rarely into the peripheral blood stream. The disease is most frequently observed in Africa, the southern part of Mexico and in Central America especially Guatemala.

Clinical Manifestations—A variety of clinical manifestations may occur the most notable being the tender subcutaneous *nodules*. These are rounded tender discolored tumors something more or less than an inch



Fig 981—*Draculus med n nris* part lly extracted (Courtesy of Maj J M Hulsey Jr MC AUS through Lt Col Hardy A Kemp MC AUS Army Medical School)

in diameter. They are freely movable and located chiefly upon the scalp. They may appear also upon the brow, the upper back and the arms. At times there is also present a generalized lichenoid dermatitis accompanied by intense *pruritus*. In a small number of instances the microfilariae invade the eye by migration from the scalp. They may then induce conjunctivitis, keratitis or iritis with resultant visual impairment or even total blindness (Blinding Filariasis).

Diagnosis and Treatment—The diagnosis is made by the demonstration of the parasites in material aspirated from the nodules. If this fails a biopsy will reveal their presence.

The only successful *therapy* is the complete and early removal of the nodules. A careful search is made for incipient lesions after all obvious areas have been extirpated.

DRACONTIASIS

Dracontiasis is caused by the guinea or dragon worm (*Dracunculus medinensis*). The important endemic area is in the Nile valley but the infestation is also seen on the west coast of Africa in Arabia and India. The worm is present in fur bearing animals of the United States.

The adult worm lives in the subcutaneous connective tissues. The male dies after fertilizing the female. When mature the latter migrates to the legs and feet where there is contact with water. On reaching the skin the female causes a blister which ruptures and exposes the head of the worm. The gravid uterus extrudes through the lesion and discharges larvae which become actively motile in water. They are ingested by their intermediate host the crustacean *Cyclops* in whose intestines they metamorphose into the infective forms. Man becomes infected by drinking water that has been contaminated. The larvae are activated by the gastric juice, penetrate the wall of the intestines and somewhere in the body develop to maturity within a month or more, forming ulcerating skin lesions. Associated with the dermatosis there may be fever, prostration, a generalized urticaria and an eosinophilia.

Treatment—See p. 3325

CHAPTER 140

THE REGUMENTARY SYSTEM ALLERGY

Diagnostic Skin Allergies
Clinical Skin Allergies
Eczema
Contact Dermatitis
Drug Eruptions
Atopic Dermatitis
Urticaria
Angioneurotic Edema
Physical Allergy
Bacterial Allergies of the Skin

ALLERGY is a term introduced by von Pirquet who intended it to signify that a living tissue or organism had acquired an altered capacity to react to a specific substance. The allergic tissue on accidental or deliberate exposure to the specific allergen responds in a manner different from normal or non-allergic tissue. The normal or average response is called *normergy*; an increased response indicative of heightened sensitivity is *hyperergy* or *hypersensitivity*; a diminished response indicating subnormal sensitivity is *hypoergy* or *hyposensitivity*; a total absence of response is *anergy*.

The more complete discussion of the general principles and broader implications of allergy beyond the cutaneous expressions constitutes a separate section in these volumes to which the reader would do well to refer (p 547).

DIAGNOSTIC SKIN ALLERGIES

The reaction of the skin to exposure to an allergen is deliberately investigated for diagnostic purposes. The responses to *pollens* (p 559) *digestants* (p 562) *contactual substances* (p 3330) *biologicals* (p 83) *drugs* (p 3335) and *toxins* (p 78) illustrate the utilization of the cutaneous response as an indicator of altered sensitivity in other shock organs. Perhaps the widest employment of the skin test is in the investigation of the specific infectious diseases where a bacterial allergy has been established. Thus a considerable amount of information is available relative to the *tuberculin test* (p 262) the *Frei test* for lymphogranuloma venereum (p 472) the *Ducrey test* for chancroid (p 289) the *Foshay test* for tularemia (p 325) the *brucellergen test* for undulant fever (p 317) the *coccidioidin test* for coccidiosis (p 3314) and the *trichinella test* for trichinosis (p 541). In these examples the cutaneous reaction of hypersensitivity denotes that the patient is or has been infected with the specific microorganism.

CLINICAL SKIN ALLERGIES

The skin reacts also in its own right as a shock organ to allergens that are unintentionally introduced. As a result the practitioner encoun-

ters a variety of dermatoses of allergic origin. The discussion of these interesting and often baffling difficulties constitutes the material of the present section.

ECZEMA

The term 'eczema' literally means to boil out. As used by the dermatologists it is applied to 'certain superficial inflammatory skin diseases not due to micro organisms which at some time in their evolution present macroscopic or microscopic evidence of intra epidermal vesicle formation (blister or bleb)'. In the present exposition an effort will be made to avoid this definition of eczema since it is a concept that is more apt to be confusing than helpful in the treatment of the patient.

Other than the dermatophytoses, the majority of skin conditions grouped as eczematous probably are manifestations of *contact dermatitis* or *atopic dermatitis*. In the former instance the dermatosis results from direct application of the offending substance; in the latter the skin reacts to noxious agents carried in the blood stream. This viewpoint has more than academic interest since it focuses the attention of the practitioner upon the etiologic agency rather than the meaningless description of the skin reaction. His therapy is then directed at discovering and eliminating the cause rather than seeking local cures for the lesion.

CONTACT DERMATITIS (ECZEMA OCCUPATIONAL DERMATITIS DERMATITIS VENENATA DERMATITIS ECZEMATOSA)

Contact dermatitis is a manifestation of cutaneous allergy (p. 547) resulting from an induced state of hypersensitivity of the epidermis to some substance with which it has come into exogenous impingement. The eruption may be *acute*, *subacute* or *chronic*. It may affect the skin, the accessible mucous membranes or both. Depending upon the site of contact between the offending agent and the cutaneous surface the distribution of the dermatosis may be *localized*, *generalized* or rarely *universal*. It may occur in either sex at any age.

Etiology—In dermatitis venenata the allergen reaches the skin from without and the shock tissue is the *epidermis*. The excitant is usually a simple or complex chemical of non protein character in contrast to the atopies where the allergen is protein or a protein derivative.

There is no explanation for the susceptibility of some persons to this type of eruption unless it is some inherent factor of unknown nature. It is difficult to understand why a certain perfume or dye can be tolerated by a large number of people who use it frequently over long periods of time while a small number develop an allergic hypersensitivity with consequent dermatitis. Circulating antibodies (respons) are not demonstrable in the blood stream or by means of the passive transfer technique of Prausnitz-Kustner (p. 559).

The number of agents capable of causing a contact dermatitis is legion. Most industrial (occupational) dermatoses fall into this classification as do usually the eruptions produced by cosmetics, insecticides, metals, leathers, fabrics, plants and topical medications. Sensitization to a particular substance may appear very soon after exposure or years of exposure may be required before evidences of cutaneous hypersensitivity are first evinced.

Obviously the most important single factor in etiology is the opportunity for exposure to a sensitizing agent. The occupation and avocation of the individual, the clothing, the personal habits of hygiene and the environment in which he works, plays and lives require close scrutiny to discover the immediate and specific etiologic agent.

The Eruption—The commonest manifestation of contact dermatitis as seen by the practitioner is the eruption of *poison ivy*

In the *acute stage of contact dermatitis* the skin is red and swollen. Vesicles appear to a greater or lesser degree. The patient complains of heat



Fig 982.—A Atopic neurodermatitis (eczema) in adult male. Probably psychogenic. B Atopic dermatitis (eczema) in child. Of digestive etiology. C Contactual atopy from cosmetic (lipstick). D Contactual atopy from wearing apparel (hatband). E Contactual atopy from occupational contact with cosmetic (hair-dye) in beauty shop operator. F Contactual dermatitis from drug (sulfathiazole ointment).

and burning of the involved area and there is usually moderate to severe itching. In the *subacute phase* vesiculation, redness and swelling become less prominent and the involved skin is thickened and scaly. When the condition becomes *chronic* and the disease is of long duration the skin becomes infiltrated, thickened, dry and scaly (lichenification).

Distribution—The distribution of a contact dermatitis is dependent upon the nature of the offending agent. Patients with an *occupational type* of dermatitis usually exhibit an eruption of the *hands forearms and face*. The child wearing socks who wanders in a patch of poison ivy shows a *leg lesion* that is situated between the top of the sock and the lower border of the underclothes. A bandlike eruption of the *wrist* suggests irritation from a bracelet or a wrist strap. A rash on the *ear lobes* may be due to ear rings. An eruption *behind the ears along the temples* and over the *nasal bridge* should make one suspect an irritation due to spectacle frames. Lesions of the *scalp* suggest hair dyes, hair tonics or shampoos. *Axillary eczema* may be due to anhydrotics, deodorants, dress shields or dress dyes. *Facial lesions* are usually due to soaps or cosmetics and the *eyelids* are most susceptible to nail polish. *Neck lesions* are often due to the dye of a fur piece. *Body eruptions* are due to clothes. Nylon corsets or laundry soap used in washing clothes. The *penis* may be sensitive to the rubber of a condom or the secretions or contraceptive of the female. A rash in the region of the *trouser pocket* may be due to the sulfur of the matches carried therein and a rash on the legs may be caused by rubber or nylon stockings.

The Patch Test—In contact dermatitis the patch test may be helpful, misleading or harmful. It is of value when it is positive and clearly reveals the offending substance. It is misleading when it is negative on one area (such as the abdomen or arm) when another area is definitely sensitized (as the eyelids to nail polish). It is both misleading and harmful when there are multiple sensitivities and the positive reactions cause an exacerbation of all lesions.

Differential Diagnosis—The diagnosis of contact dermatitis should be suspected in dealing with any eruption that is characterized by the formation of vesicles (p. 3334) and any type of strictly localized persistent skin affection that fails to respond to simple local therapy.

A number of dermatoses may at one time or other in their course bear a striking resemblance to dermatitis venenata. On morphologic grounds alone even the expert will have difficulty in distinguishing them.

Local Treatment—The successful treatment of contact dermatitis requires the *discovery of the offending agent* (p. 3332) and its elimination. Pending the outcome of diagnostic investigation the simplest forms of local treatment are applied. The practitioner should constantly bear in mind the possibility that the patient will exhibit multiple sensitivity and that the use of the simplest chemicals may superimpose a treatment dermatitis. Introduction of antihistamine agents, notably pyribenzamine in 50 mg. tablets four times daily, affords striking symptomatic relief (p. 505).

Acute Phase—In the acute phase *soothing wet dressings of cold milk* are very useful. With extensive eruptions the *starch bath* (p. 3133) affords great relief without risk. If it is inconvenient to employ the wet dressings or the bath, the *calamine shake lotion* (p. 3115) is painted on the lesion. If itching is severe, 0.5 per cent *menthol* or 0.25 per cent *phenol* (p. 3137) is added cautiously for symptomatic relief. In this phase of the disturbance *soap water pastes and ointments are strictly to be avoided*.

Subacute Phase—When the eruption becomes less acute *soothing pastes* such as the *Issar formula* (p. 3132) or *ointment* may be applied. The

latter may contain 3 to 5 per cent ethyl aminobenzoate (p 3114) Liquor Carbonis Detergens (3 to 5 per cent) or Oil of Cade (1 to 3 per cent) if the itching is severe

Chronic Phase—In the chronic phases with lichenification *stronger*

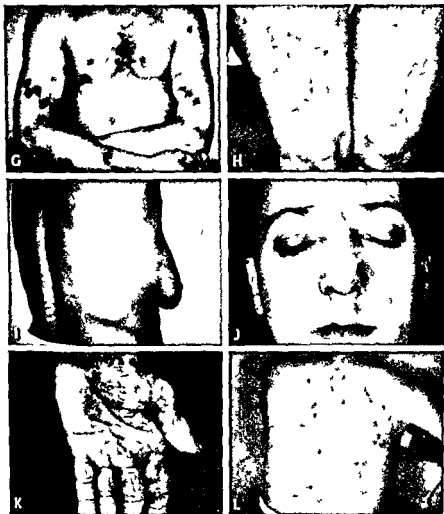


Fig 983—G Drug atopy Dermatitis medicamentosa from phenolphthalein taken as cathartic H Drug atopy Dermatitis medicamentosa from phenobarbital taken as sedative I Drug atopy Dermatitis medicamentosa from arsenic injections for syphilis J Contactual atopy (occupational) From refrigerating gas K Contactual atopy (occupational) From tar L Trichophytid from ringworm of feet

tar concentrations (p 3126) may be used as well as *salicylic acid* (p 3126) and *ammoniated mercury* in ointment form With persistence of symptoms the patient should be referred to the *specialist consultant* for his opinion and for *roentgen therapy*

Prophylaxis of Plant Dermatitis—For the prevention of contact der

DIFFERENTIAL DIAGNOSIS OF

Local Vesicular, Bullous and Pustular Dermatoses

Vesicular bullous and pustular dermatoses are rarely seen without complicating scabs, crusts and exfoliations which may obscure the nature of the underlying lesion

See also Generalized Vesicular and Pustular Eruptions p 422

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|---------------------------------|--|
| Acne varioliformis | Chronic remittent dermatosis Crops of papulo-pustules of face Pitted scars after healing (p 3356) |
| Acne vulgaris | Chronic remittent dermatosis of face and trunk In adolescents Characterized by comedones papules pustules oiliness and cystic or pitted lesions (p 3358) |
| Anthrax | Malignant pustules Halo of vesicles Isolate gram positive spore bearing bacillus by smear and culture (p 292) Start intensive antibiotic therapy |
| Bullous impetigo | With exfoliative dermatitis in infants and newborn A malignant staphylococcal infection (p 3252) Start intensive penicillin therapy |
| Chilblain | Frostbite followed by vesiculation (p 3173) Consider use of anticoagulants |
| Dermatitis herpetiformis | Unusual bilateral and symmetrical dermatosis with grouped erythematous patches papules vesicles pustules bullae and pigmentation (p 3371) |
| Dermatitis papularis capillitii | Papulopustular lesion of neck Especially seen in Negroes On healing leaves a hard keloid (p 3255) |
| Dermatomycoses | Pustulation with granuloma formation and ulceration Identify pathogen by smear and culture (p 3293) |
| Epidermolysis bullosa | Rare congenital anomaly Characterized by bulla formation at site of pressure or trauma (p 3151) |
| Folliculitis decalvans | Circumscribed areas of pustulation of scalp followed by scarring and permanent alopecia (p 3442) Note effects of vitamin A |
| Furuncle | The simple boil Usually a local staphylococcal invasion Try local penicillin therapy |
| Helminthiasis | Vesiculation of skin at site of entry of uncinariæ and schistosomes Identify invading parasite (p 537) |
| Herpes simplex or Zoster | Fig 961 p 3289 |
| Impetigo contagiosa | Local staphylococcal infection of face With formation of vesicles pustules and crusts (p 3251) Try local treatment with sulfathiazole or penicillin |
| Pneumococemia | With herpes of lip |
| Pustular folliculitis | Staphylococcal infection of follicular apertures (p 3249) Try local therapy with sulfathiazole or penicillin |

| | |
|------------------|--|
| Scabies | Vesiculation and pustule formation surrounding burrow containing <i>acarus</i> . Note itching. Identify parasite (p. 3180). |
| Sycosis vulgaris | Chronic staphylococcal inflammation of hair follicles. Particularly of male beard (p. 3249). Try local treatment with sulfathiazole or penicillin. |
| Syphilis | Follicular and pustular syphilids. Check dark field in addition to serology (p. 336). |
| Vaccinia | Local take with pustulation and scar formation. |

matitis due to plants such as *poison ivy* or *poison oak* the affected area is rubbed vigorously with 95 per cent alcohol the tops of the vesicles are removed the serum is wiped away and 10 per cent aqueous tannic acid solution is applied. One half hour later the tannic acid solution is painted on again. The patient is instructed to repeat this treatment twice daily after uncapping the vesicles each time. After the acute phase has subsided or in a free interval prophylactic desensitization may be accomplished by two or three intramuscular injections of poison ivy and poison oak antigen given in doses of 1 cc. at 24 or 48 hour intervals.

DRUG ERUPTIONS (DERMATITIS MEDICAMENTOSA)

A drug eruption is an abnormal response on the part of the skin and/or mucous membranes to a medicament which reaches it through the blood stream. In the majority of instances drug eruptions are a manifestation of hypersensitivity but they may be due to retention and cumulation.

Drug eruptions constitute one of the manifestations of cutaneous allergy. They differ from contact dermatitis in that they are produced by therapeutic substances administered enterally or parenterally rather than by external application. They differ from atopic dermatitis in that the sufferer may be of any age there is rarely a family history of other manifestations of allergy and there are no other disturbances in shock organs such as the nasal mucous membrane or the bronchial musculature.

Drug eruptions vary in their gravity from the merely annoying pimples of iodine or bromide to the often fatal manifestations of dermatitis exfoliativa from gold or arsenical poisoning. They are more often manifestations of idiosyncrasy than overdosage. Since their occurrence is unpredictable the practitioner should not be held culpable when they appear in patients under his treatment. However the alert physician may often detect evidences of drug eruptions in their incipency when immediate cessation of the drug may prevent a serious involvement.

THE ERUPTION

The eruptions produced by drugs may be many and varied. They are recognized only when the possibility is carefully considered. The practitioner who makes it a rule to inquire into the possibility of the presence of a dermatitis medicamentosa in every cutaneous rash cannot but be struck by the high incidence of this dermatosis.

After the first or *sensitizing* dose of the drug a subsequent or *eliciting* dose must be given before the eruption appears. The time interval (in

cubation period) between the sensitizing and eliciting doses may vary from a few hours to many years. The eruption may therefore appear

DIFFERENTIAL DIAGNOSIS OF

The Commoner Generalized Erythematous or Scarletiform Rashes

Generalized erythematous eruptions are most often of infectious origin with the notable exception of dermatitis medicamentosa. In childhood the practitioner is mainly concerned with differentiating scarlet fever, rubella and infectious mononucleosis. In the adult, the diagnostic dilemma becomes more difficult since secondary syphilis and drug rashes require additional consideration. A history of previous scarlet fever or rubella narrows the possibilities since second attacks of either of these infections are rarely if ever observed. Laboratory findings may add confusion since infectious mononucleosis is frequently accompanied by a false positive Wassermann reaction. Unless the patient gives a clear cut history of exposure to syphilis or presents other manifestations of venereal infection, a definite opinion should be deferred and treatment may be withheld until the course of the Wassermann test can be followed during the fading of the heterophile reaction and the disappearance of mononuclear cells from the blood. Finally it must not under any circumstance be forgotten that common drugs such as the analgesics, quinine, salicylates and sulfonamides may produce scarlatiniform eruptions in those who suffer from an idiosyncrasy. When the drug rash is accompanied by a drug fever, the resemblance to an infectious disease is the more closely simulated.

DIAGNOSTIC FEATURES

| | |
|--------------------------|--|
| Dermatitis Medicamentosa | A drug eruption following analgesics, quinine, salicylates, arsenicals, phenolphthalein, sulfonamides, etc. (p. 3335) |
| Fourth Disease | Occurs in infancy. Diffuse erythema of face, body and extremities (p. 418). No constitutional symptoms or lymphadenopathy. Schultz-Charlton test negative. |
| Erythromelalgia | Vasomotor neurosis of young and middle aged females. Episodes of pain, redness and swelling of hands and/or feet (p. 1002). |
| Infectious Mononucleosis | Generalized lymphadenopathy (glandular fever) with characteristic hemogram (p. 466), positive heterophile reaction (p. 468) and false positive Wassermann (p. 468). |
| Rubella | May produce scarlatiniform rash. Posterior cervical lymphadenopathy but mild constitutional symptoms, rapid evolution and involution of eruption and negative Schultz-Charlton (p. 179). |
| Scarlet Fever | Acute erythrogenic streptococcal infection with fever, sore throat and vomiting. Initial eruption punctate followed by diffuse erythema and, later, desquamation. Strawberry tongue, Schultz-Charlton blanching reaction (p. 179). |
| Syphilis | Secondary syphilid with positive darkfield and serology (p. 337). |

after a second dose or it may not do so until hundreds of doses have been taken without apparent ill effect.

The manner of entry of the drug into the blood stream does not alter its capacity to elicit the cutaneous eruption. A patient who has had a dermatitis after intravenous administration of *arsphenamine* will have a recurrence of the dermatitis from an *arsphenamine* derivative (*bismarsen*) given intramuscularly or from the use of *aldarson*e given vaginally for a trichomonal infection.

Clinical Course of Drug Eruption.—Drugs can produce many different kinds of eruptions and also may have untoward effects upon other organs. *Visceral reactions* may occur along with or independent of lesions in the skin. *Fever, chills, malaise* and *arthralgia* are fairly common concomitants of certain types of drug eruption. Some drugs produce *cerebral syndromes* (hemorrhagic encephalopathy), *peripheral neuropathy*, *blood dyscrasias* (thrombocytopenic purpura, agranulocytosis and aplastic anemia), *hepatitis* with jaundice and *atrophy of the optic nerve*.

In general, drug eruptions are characterized by abruptness of outbreak, a tendency to generalized and symmetrical distribution and itching. There are, however, many exceptions to these rules.

Drug eruptions may have morphological manifestations that are so variegated that it is impossible accurately to diagnose them or differentiate them from other dermatoses without knowledge of the history. Thus they may be *morbilliform*, *scarlatiniform*, *erythematous*, *bullous*, *acneiform*, *fungating*, *nodular*, *pigmented*, *keratotic*, *urticarial*, *purpuric*, *vesicular* or *exfoliating*.

Generalized Morbilliform and Scarlatiniform Eruptions.—Eruptions of general distribution usually appear suddenly. They are often preceded by a short period of prodromal fever with constitutional manifestations such as nausea, vomiting, headache and malaise. These eruptions usually involve the trunk and extremities most heavily. Less often the face is included though it is by no means exempt.

Generalized eruptions usually reach the stage of full development in twenty-four to seventy-two hours and then involute with a fine scaling but no generalized exfoliation. Constitutional symptoms such as arthralgia, headache, nausea and vomiting accompany the height of the rash. The fever may rise to as high as 103° or even 105° F.

The *morbilliform eruption* begins as macules which later become slightly papular resembling measles. With the *scarlatiniform eruptions* there is an initial macular erythema with rapid coalescence of the lesions and the formation of a diffuse red rash. Morbilliform and scarlatiniform eruptions occur with *sulfonamide*, *barbiturate*, *gold* and *arsphenamine*. In the latter instance the eruption has been called the *erythema of the ninth day* though it may appear anywhere between the seventh and the twelfth days following the administration of the first dose. There must, however, be a second or eliciting dose before the eruption appears. With *sulfonamides* the eruption usually appears from the fifth to the ninth day after the commencement of therapy. The sulfonamide eruption is of considerable importance since the drug is frequently given for the relief of a febrile condition. The appearance of the eruption and the recrudescence of the fever may suggest an exacerbation of the original disease or the onset of a complication. If a drug eruption is not suspected an error in diagnosis attributing the new symptom to the fundamental infection may lead the

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DIAGNOSTIC FEATURES

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after a second dose or it may not do so until hundreds of doses have been taken without apparent ill effect

Keratosis—Keratosis arises from the prolonged administration of inorganic arsenic. They are of importance as precancerous lesions (p 3209) and occur most frequently on the hands.

Urticaria and Angioneurotic Edema—Urticaria and angioneurotic edema may develop following the administration of *aminopyrine* *antipyrine* *atropine* *barbiturate* *bromide* *iodide* *quinine* *phenolphthalein* *salicylate* and *sulfonamide*. These lesions have nuisance value alone when they involve the skin. However an angioneurotic edema (p 3349) of the mouth soft palate or larynx may obstruct respiration and produce suffocation and death if prompt and heroic relief is not at hand.

Purpura—Erythema results from drugs that injure the capillaries such as acetylsalicylic acid or those which produce a thrombocytopenia (*arsphenamines* and *aminopyrine*). The latter disturbance is of the greatest gravity and especially when accompanied by depression of the formation of the red and white blood cells frequently proves fatal.

Exfoliative Dermatitis—Exfoliative dermatitis constitutes the most serious of the drug eruptions. Its manifestations and management are elsewhere described (p 3383).

Miscellaneous Eruptions—It is important to recognize that certain more or less characteristic eruptions may be elicited by drugs. They have been known to bring on *psoriasis rosea* (p 3410) *lichenoid eruptions* (p 3389) *erythema multiforme* (p 3374) *erythema nodosum* (p 3377) *stomatitis* (p 1607) and *exfoliative dermatitis* (p 3383). These eruptions cannot be distinguished on morphologic grounds from similar lesions of other causation. A fuller discussion of these conditions will be found in the sections referred to.

DRUGS FREQUENTLY USED AND THEIR COMMONER UNFAVORABLE SKIN REACTIONS

Aminopyrine and Antipyrine—Morbilliform and scarlatiniform toxicoderma, urticaria, angioneurotic edema, purpura, fixed eruptions.

Arsenic (Inorganic)—Most often employed as the solution of potassium arsenite (Fowler's solution) but also absorbed accidentally in certain occupations and foods. Skin eruptions generally appear after prolonged administration and result from cumulation and retention. The commonest eruptions are a widespread scaling erythema with thickening of the skin pigmentation (melanoderma) and keratosis.

Arsphenamines (Trivalent Organic Arsenic)—Includes all the drugs used in the treatment of syphilis except tryparsamine (pentavalent arsenic). The commonest skin manifestations are morbilliform and scarlatiniform toxicodermas (Miles' erythema of the ninth day), urticaria, angioneurotic edema, psoriasis rosea like and lichen planus like (lichenoid) eruptions, purpura, fixed eruptions, vesicular eczematoid eruptions, exfoliative dermatitis.

Atropine (also *Sellekian*)—Urticaria, angioneurotic edema and scarlatiniform erythema. **Bismuthates**—Morbilliform and scarlatiniform toxicodermas, urticaria, angioneurotic edema, purpura, fixed eruptions, bullous lesions of the mouth and genitals, exfoliative dermatitis. The barbiturates may act as photosensitizers.

Bismuth—The various forms of bismuth employed principally in the treatment of syphilis are rarely productive of eruptions. Much has been written about presumed bismuth eruptions but these must be extremely exceptional instances. Most often one encounters stomatitis and rarely embolism of the gluteal artery.

Bromides—Bromides are important cause of skin eruptions usually after prolonged use and as a consequence of retention. The dermatoses include acneiform follicular eruptions, erythema nodosum like and erythema multiforme like lesions, fungating and vegetative eruptions, urticaria, morbilliform toxicoderma, bullous eruptions which may simulate pemphigus.

Dinitro Sodium—May give rise to a morbilliform toxicoderma and hypertrophic gingivitis (in epileptics).

practitioner to increase the dosage of the drug with dire and at times fatal results

Erythemas—Localized erythematous lesions are observed most often in the 'fixed' eruptions that occur with *phenolphthalein* *arsphenamines* *antipyrine* and the *barbiturates*. The lesion consists of one or more circumscribed rounded or oval patches. When the reaction becomes more intense the erythematous area may show edema and the formation of vesicles and bullae. The patches may vary from $\frac{1}{2}$ inch to several inches in diameter. When the acute reaction subsides there is usually left a residual brown to black pigmentation which fades very slowly. Many months or even years may elapse before the pigmentation completely disappears.

The erythematous lesion may be located on any part of the skin surface. The term 'fixed' derives from the fact that with each administration of the causative drug there will appear a flare up of the quiescent spot. New lesions may spring up as well but the old area always reacts. The term 'fixed' refers to topography rather than the permanency of the lesion.

Bullous Eruptions—Bullous eruptions may appear on any part of the skin surface but are usually confined to the mouth, lips and genitals. They may be present in all of these latter areas but at the same time as is seen not uncommonly with the reaction to *phenolphthalein* and the *barbiturates*. The bullae usually enlarge rapidly at first then collapse and erode leaving a raw surface. On the penis or labia the lesion may be mistaken for a syphilitic lesion.

Acneform Eruptions—Acneform or furuncle like eruptions occur most commonly from *iodide* and *bromide*. The lesions may be confined to the face but more often they are also present on the trunk and extremities. The individual lesion is a firm red papulopustule usually situated at the orifice of a follicle. It is often dome shaped rather than conical and when it becomes markedly inflamed and pustular it resembles a furuncle. Acneform drug eruptions may be difficult to differentiate from *acne vulgaris* (p. 3358). The absence of comedones and the abrupt onset of the rash favor the possibility of a dermatitis medicamentosa.

Fungating Eruptions—Fungating and vegetating eruptions often result from the prolonged administration and retention of *bromide* and *iodide*. Nurslings may absorb these drugs from mother's milk. These ugly rashes may be single or multiple. The sites of predilection are the legs, buttocks and face though they may occur in any location. Characteristically they consist of raised fleshy masses which may be flat, papillomatous, cauliflower like or necrotic. At times the surface is studded with tiny pustules so that the lesion resembles the eruptions of syphilis, tuberculosis, the deep mycoses and even cutaneous malignancy.

Photosensitivity and Drug Eruptions—Photosensitivity resulting from the administration of drugs may cause curious eruptive and pigmentary disturbances of the exposed parts. Most often the disturbance is revealed on the face, neck and the backs of the hands. In women who wear low necks the V of the chest and the forearms are also involved.

The most important drugs which act as photosensitizers are *sulfonamides* especially *sulfanilamide*, *barbiturates*, *quinine* and the *gold salts*. Patients who receive these should be warned against exposure to sunlight, ultraviolet rays and roentgen rays.

cedure since the subsequent attack may be far worse than the first. It may cause serious illness and in some instances may terminate fatally. If the primary disease is serious and the drug although essential can safely be temporarily discontinued—as in syphilis (the arsphenamines) and dangerous infections (the sulfonamides)—this procedure is permissible. If possible the substitution of another homologous drug may be practiced with the hope of lessening the chance of reaction.

The mere fact that a patient has been taking a drug or the presence of the drug in the urine does not indicate that it is the cause of an eruption. In the therapy of syphilis the appearance of an eruption usually raises the question of its relationship to the arsenical. In some cases the decision is difficult and may have to await a period of rest from the arsenical and its subsequent re-administration.

PROGNOSIS OF DRUG ERUPTIONS

Drug rashes may be minor reactions of no serious significance or they may be severe enough to cause prolonged illness and even death. Particularly dangerous is exfoliative dermatitis due to the arsphenamines, gold salts, barbiturates and sulfonamides. Fungating and gangrenous iododermas have resulted fatally in a number of instances.

Often the cutaneous eruption is accompanied by serious concomitant injury of other tissues and it may be the latter which causes a fatal outcome. This is especially true of bone marrow damage which may lead to *agranulocytosis* or *aplastic anemia*. *Hemorrhagic encephalopathy* (about 76 per cent fatalities) may co-exist with morbilliform and scarlatiniform toxicoderma due to arsphenamine. A not uncommon coincidence in the case of the arsphenamines is severe *hepatitis* with atrophy and serious *nephritis*.

The fixed eruptions are not serious but often present a difficult problem in therapeutics. When they occur in syphilis where further therapy with the arsphenamines may be vital, change to penicillin should solve the problem.

TREATMENT OF DRUG ERUPTIONS

The simpler eruptions require little or no treatment since they are generally short-lived and unaccompanied by severe systemic symptoms.

Striking symptomatic relief in dermatitis medicamentosa accompanies the administration of pyribenzamine (p. 565) in 50 mg doses. Locally the use of a *soothing and antipruritic powder, lotion or ointment* is often gratifying. For widespread eruptions if the patient's condition warrants, starch baths are useful. *The responsible drug must be stopped* and not given again. In the case of erythema of the ninth day due to arsphenamine it is generally accepted that the arsphenamine may be continued. There is only a single recorded instance in which continuance led to the production of exfoliative dermatitis. However it has been our experience that if the arsenical is given too soon after the reaction there may be a recurrence of reaction with or without reappearance of the rash. The introduction of BAL (dimercaptol) provides potent detoxification in arsenical disturbances (p. 767).

Exfoliative dermatitis is a serious reaction and in every instance the patient should be hospitalized. When due to arsphenamine *dimercaptol*

Gold Salts—Eruptions are not uncommon and are similar to those observed from the arsphenamines (p 3339) Gold may act as a photosensitizer The combination of gold injections and exposure to ultraviolet light or sunlight produces chrysiasis a pigmentation due to deposit of the salt in the cutis

Iodides—The most important are acneiform furuncle like lesions fungating and vegetating tumors purpura bullous eruptions morbilliform and scarlatiniform toxicodermas urticaria angioneurotic edema gangrenous ulcerations

Mercury—When mercury is given orally or by injection for syphilis and other diseases it is an uncommon excitant of eruptions other than stomatitis When applied to the skin it may be absorbed and provoke a widespread scaling erythema

Opium and its Derivatives—The most frequent cutaneous manifestation is generalized pruritus but actual urticaria may occur Codeine seldom causes untoward effects in the skin

Penicillin—Penicillin eruptions may occur after local or systemic use Most often they are erythematous urticarial or vesicular

Phenolphthalein—This drug is the active ingredient of many proprietary laxatives It is said also to be present in pink icing of cakes and even in these small amounts may cause a reaction in sensitive persons It is a common cause of cutaneous lesions The important ones are fixed eruptions bullous lesions of the mouth and genitals erythema multiforme like lesions urticaria eczematous eruptions

Quinine—Quinine is present in many hair tonics and also in proprietary headache and cold remedies Quinine is peculiar in that it can excite a contact dermatitis by local application and also eczematous eruption by ingestion In persons who have once had a contact dermatitis from quinine the eruption may be re-elicited by ingestion of the drug The commoner lesions caused by quinine are eczematous eruptions urticaria angioneurotic edema morbilliform and scarlatiniform toxicodermas bullous eruptions purpura fixed eruptions Quinine may act as a photosensitizer

Salicylates—The salicylates especially aspirin are undoubtedly the most frequently self administered analgesic They may provoke urticaria angioneurotic edema erythema multiforme-like and erythema nodosum like eruptions morbilliform and scarlatiniform toxicodermas bullous lesions purpura

Silver—The administration of inorganic silver (silver nitrate) by ingestion is extremely rare today However the use of this salt as well as colloidal silver preparations topically may lead to *argyria* (p 758)

Sulfadiazine—Eruptions have been seen in about 2 per cent of cases They are either morbilliform or erythema nodosum like

Sulfaguanidine—Eruptions are rarely seen only two instances having been recorded both being morbilliform toxicodermas

Sulfanilamide—Causes eruptions in about 2 per cent of cases The lesions observed include morbilliform and scarlatiniform toxicodermas vesicular and bullous eruptions urticaria purpura fixed eruptions stomatitis exfoliative dermatitis It is an important photosensitizer the exposed areas developing a maculopapular eruption edema and bullae

Sulfapyridine—Sulfapyridine has an incidence of drug eruption in 2 per cent of cases The manifestations are generally similar to those noted with sulfanilamide (p 88)

Sulfathiazole—Eruptions are encountered in 5 per cent of cases They are chiefly erythema nodosum like morbilliform and urticarial In about 4 per cent there is seen a conjunctival and scleral injection which may accompany a skin eruption or may exist alone

Tryparsamide—A pentavalent arsenical used in the treatment of neurosyphilis It rarely causes dermatitis but fixed eruptions and urticaria have been observed Its most important toxic effect is atrophy of the optic nerve

DIAGNOSIS OF DRUG ERUPTIONS

Every history should include an account of the drugs the patient has been taking The patient should be questioned specifically as to the use of patent medicines laxatives headache and cold remedies analgesics for menstrual pain hangover cures etc

The proof of the presence of a drug eruption requires the physician to stop the drug allow the eruption to clear and then reproduce the eruption by administering the drug again This may be a hazardous pro

urticaria strophulus lichen urticatus disseminated neurodermatitis or flexural eczema and in adult life as *disseminated neurodermatitis*

Infantile Eczema.—Infantile eczema usually appears in the first six months of life and only rarely after the second year. Most of the afflicted clear up spontaneously by their second or third birthdays.

The eruption of infantile eczema consists of *erythematous patches* with *papules vesicles* and some *scaling*. Oozing and crusting may be absent or very marked. *Itching* is usually very intense producing great discomfort. The lesion most commonly involves the head neck and extremities though it may become widespread and even generalized. Isolated patches are often seen on the cheeks.

The condition occurs in both sexes and is prevalent from infancy up to the age of thirty. Although scratch and intradermal tests and passive transfer studies may reveal many positive reactions to food and other allergens the specific *etiologic agent* often remains elusive. Removal of the reacting substance only too frequently leads to no material improvement in the condition. However in occasional instances especially in infancy *dramatic response* ensues. The most important allergens are foods but environmental contactual and inhalant allergens may also play an important role (orris root animal danders silk wool dust plants etc.)

The skin in atopic dermatitis is extremely vulnerable to a great many ordinarily innocuous factors. Thus *heat cold soap and excessive changes in temperature* may cause aggravation of the disease. *Nervous strain and emotional upsets* have an untoward effect possibly by lowering the threshold for itching. Some investigators believe that the psychogenic factor is of the utmost significance.

Prurigo Mitis.—Prurigo mitis is seen later in childhood. The eruption begins as an *urticarial wheal* which upon being scratched becomes red dened crusted and excoriated. *Fleshy papules* are formed which are isolated and discrete and present in small or great numbers.

Prurigo mitis is chiefly distributed upon the *extensor aspects* of the upper and lower *extremities* but lesions may also be present upon the trunk and face.

Disseminated Neurodermatitis.—Disseminated neurodermatitis occurs in childhood and adolescence. The lesion is a raised *plaque* of thickened skin having a cross hatched surface which usually shows a slight amount of scaling. The eruption may be skin colored brownish or grayish.

In *childhood* the eruption of disseminated neurodermatitis has a predilection for the natural skin folds particularly the elbows the bend of the knee and the neck. Most of the changes result from prolonged scratching and rubbing and conform to the clinical picture of lichenification (p 3168) with secondary excoriations crustings and pyogenic lesions (p 3248).

The *adult form* of disseminated neurodermatitis is more apt to involve the scalp face eyelids neck and upper chest. This condition tends gradually to disappear before the fourth decade of life.

The present concept is that these different morphologic types are phases of atopic dermatitis. The manifestation produced is conditioned largely by the physiological alterations in the skin at the various age periods. Thus the disease may begin in infancy and continue without remission but with alteration in appearance into adult life. On the other hand after the infantile phase clears up it may never reappear.

(BAL) is given by intramuscular injection as elsewhere described. Undoubtedly the most valuable therapy is an *intravenous drip* of 5 per cent dextrose solution giving 500 to 1500 cc daily according to indication. Injections of *liver extract* have been recommended highly. Local treatment consists of daily starch baths and the application of calamine liniment. In cases of exfoliative dermatitis due to arsphenamine (or any other drug) the drug should never again be administered in any form. We have seen two patients die after their second attack of exfoliative dermatitis due to arsphenamine. The patient must be warned against allowing a physician ever to give him any one of the trivalent arsphenamines.

The *iododermas* and *bromodermas* (p 3340) are characterized by their chronicity because of the retention of these drugs by the tissues. Efforts are therefore directed to hastening their elimination, and this is usually achieved by the administration of *chlorides*. The chloride ion replaces the iodide and bromide ions and thus speeds excretion. In milder cases tablets of sodium chloride 1 gm each are used in doses of 3 gm four times daily. Highly salted foods may also be added to the diet. Where the eruption is severe slow intravenous drip of 1000 to 1500 cc of normal saline may be given until improvement occurs then less frequently and in smaller amounts. This is particularly important in severe bromide intoxication with cerebral symptoms. Continuous gastric drainage to remove the drug from the stomach has also been recommended for bromide intoxication. One must also be careful in *iododerma* to eliminate iodized salt as a possible contributory factor. In the tuberous and fungating forms roentgen ray therapy often hastens the involution of the lesions.

Patients receiving photosensitizing drugs should not be exposed to ultraviolet light or roentgen rays and should be warned to avoid direct sunlight.

ATOPIC DERMATITIS

Atopic dermatitis comprises a number of distinct cutaneous disturbances which occur in individuals who have other evidences of hypersensitivity. The common denominators of the atopic conditions include a high familial incidence of some form of allergy (asthma hay fever) an abnormal number of positive scratch tests to protein materials the presence of positive passive transfer tests (Prausnitz Kustner) eosinophilia and in addition to the atopic dermatitis a tendency to develop at some later time other manifestations of allergy. In all of these respects the patient with atopic dermatitis differs from the sufferer from contact dermatitis or drug dermatitis. In the latter conditions all other allergic phenomena are usually conspicuously absent.

THE ERUPTION

Atopic dermatitis includes a number of distinct and related conditions. These skin manifestations occur almost exclusively from the period of infancy up to about the thirtieth year beyond which they are only rarely encountered. In their various aspects they have been described under many different and confusing names. In infancy the condition is best known as *infantile eczema* in childhood and adolescence as *prurigo mitis* at

urticaria strophulus lichen urticatus disseminated neurodermatitis or *flexural eczema* and in adult life as *disseminated neurodermatitis*

Infantile Eczema—Infantile eczema usually appears in the first six months of life and only rarely after the second year. Most of the afflicted clear up spontaneously by their second or third birthday.

The eruption of infantile eczema consists of *erythematous patches* with *papules vesicles* and some *scaling*. Oozing and crusting may be absent or very marked. *Itching* is usually very intense producing great discomfort. The lesion most commonly involves the head, neck, and extremities, though it may become widespread and even generalized. Isolated patches are often seen on the cheeks.

The condition occurs in both sexes and is prevalent from infancy up to the age of thirty. Although scratch and intradermal tests and passive transfer studies may reveal many positive reactions to food and other allergens, the specific *etiologic agent* often remains elusive. Removal of the reacting substance only too frequently leads to no material improvement in the condition. However, in occasional instances, especially in infancy, dramatic response ensues. The most important allergens are foods, but environmental contactual and inhalant allergens may also play an important role (orris root, animal danders, silk, wool, dust, plants, etc.).

The skin in atopic dermatitis is extremely vulnerable to a great many ordinarily innocuous factors. Thus *heat*, *cold*, *soap*, and *excessive changes in temperature* may cause aggravation of the disease. *Nervous strain* and *emotional upsets* have an untoward effect, possibly by lowering the threshold for itching. Some investigators believe that the psychogenic factor is of the utmost significance.

Prurigo Mitis—Prurigo mitis is seen later in childhood. The eruption begins as an *urticarial wheal* which, upon being scratched, becomes red, dened, crusted, and excoriated. Fleshy papules are formed which are isolated and discrete and present in small or great numbers.

Prurigo mitis is chiefly distributed upon the *extensor aspects* of the upper and lower *extremities*, but lesions may also be present upon the trunk and face.

Disseminated Neurodermatitis—Disseminated neurodermatitis occurs in childhood and adolescence. The lesion is a raised *plaque* of thickened skin, having a cross-hatched surface which usually shows a slight amount of scaling. The eruption may be skin-colored, brownish, or grayish.

In *childhood*, the eruption of disseminated neurodermatitis has a predilection for the *natural skin folds*, particularly the *elbows*, the *bend of the knee*, and the *neck*. Most of the changes result from prolonged scratching and rubbing and conform to the clinical picture of lichenification (p 3168) with secondary excoriations, crustings, and pyogenic lesions (p 3248).

The *adult form* of disseminated neurodermatitis is more apt to involve the *scalp*, *face*, *eyelids*, *neck*, and *upper chest*. This condition tends gradually to disappear before the fourth decade of life.

The present concept is that these different morphologic types are phases of atopic dermatitis. The manifestation produced is conditioned largely by the physiological alterations in the skin at the various age periods. Thus the disease may begin in infancy and continue without remission, but with alteration in appearance into adult life. On the other hand, after the infantile phase clears up, it may never reappear.

Fortunately, in the great majority of cases the disease tends eventually to spontaneous cure. It rarely persists up to or past the age of thirty. Those extremely rare cases which do persist are found to be resistant to therapy. A certain number of these individuals will develop other atopic manifestations (asthma hay fever) after recovery from the cutaneous disorder. Asthma or hay fever may precede the cutaneous disturbance or it may exist along with it.

DIAGNOSIS

The diagnosis is generally simple: the age of the patient, the distribution and character of the eruption, the family history of atopy and the presence of positive scratch and intradermal tests constituting vital evidence. The absence of positive patch tests of a plausible contact factor and of the other usual criteria of dermatitis venenata (p 3330) are of value in a negative sense.

SUGGESTED ADDITIONAL EXAMINATION

Patients with atopic dermatitis commonly have (1) a family history of allergy (2) positive skin tests to the common pollens and digestants (milk eggs and wheat) (3) other evidences of allergy such as *vasomotor rhinitis* or *asthma*.

TREATMENT

The management of the infant with eczema requires attention to the diet, the environment and the local condition. *Dietary control* is of great importance in the infant but later in life assumes less significance. It may be pursued (1) by gradual elimination of one item of food after another from the diet (2) by the use of special elimination diets (Rowe Cobb) of low antigenic index (p 683), or (3) by feeding a specific diet based upon the findings in scratch tests. Cow's milk (p 634) is a common allergen and in its place soybean emulsion, goat's milk or evaporated milk may be introduced. *Cane sugar* can replace the usual carbohydrate adjuvants, *ascorbic acid*, the citrus fruits and *drisdol*, the cod liver oil.

Environmental control includes the use of a bedroom without rugs, drapes or curtains and the elimination of house dust as completely as possible. Pillows must be discarded and also garments of wool and silk. Only linen or cotton should be worn. The mattress is enclosed in a non-allergenic cover. Animals, plants and toys covered with fur or cloth are removed. Soap should never be used on the infant's skin, baths being given with corn starch. Mineral oil is used as an emollient and cleanser and the so-called antiseptic baby oils are avoided. Where itching is intense the *tar bath* (p 3133) is given. Crusts and scales are removed with olive or mineral oil or boric acid compresses. Dusting powders, zinc stearate and unscented talcum may be used liberally.

The most valuable remedy in the treatment of infantile eczema is *tar* (p 3131). If there is markedly acute inflammation, *soothing applications* should first be used. These include wet dressings of Boric Acid Calamine Liniment, Calamine Lotion or Paste of Zinc Oxide. To these may be added *antipruritics* as 0.25 to 0.5 per cent Phenol or 0.25 to 0.5 per cent Menthol. Liquor carbonis detergens in 2 to 5 per cent strength may be added as the inflammation subsides. When the acute phase has been controlled, crude coal tar 1 to 3 per cent in ointment or paste of zinc ox may

be used. For the scalp the coal tar should be incorporated in cold cream or equal parts of lanolin and petrolatum. It is important that the ointments be applied evenly and covered with soft materials (pillowcase bed sheet linen) and that they be removed once daily by cleansing with olive or mineral oil.

In childhood adolescence and adult life the value of elimination of allergens as in infantile eczema is small. Nevertheless it is desirable to do as much as possible in this direction in the removal of food inhalant and environmental allergens. Topical therapy is of distinct value chiefly the use of the tars as advocated for infantile eczema. Every effort should be made to keep the patient from scratching by the use of local anti pruritic remedies avoidance of noxious influences (soap water cosmetics) on the skin and administration of sedatives (phenobarbital bromides) where necessary. Ultraviolet light therapy is beneficial in many cases. Auto hemotherapy giving 5 to 10 cc of blood twice weekly seems to have favorable influence on many individuals. Roentgen therapy is excellent for clearing up areas intractable to other forms of treatment.

The avoidance of emotional stress is recommended. Frequently a change of environment as merely leaving the home and entering the hospital will produce remarkable improvement.

The introduction of anti histamine agents has greatly improved symptomatic treatment (p 565). Pyribenzamine or benadryl in 50 to 100 mg doses four times daily provides relief from pruritus particularly.

URTICARIA (HIVES)

Urticaria is an acute or chronic disease due to many diverse causes and characterized by the appearance of transient pruritic wheals. The urticarial eruption may be the sole manifestation of some fleeting and unimportant toxic action upon the skin but it may represent the cutaneous symptomatic response to a serious underlying visceral disease.

Pathogenesis.—The noxious agent reaches the capillaries of the cutis in the circulating blood and damages the endothelial cells of the vessels. As a result, there is marked dilatation of the vessels especially in the papillary layer and impairment of their functional if not an anatomical integrity. Transudation of serum and white blood cells into the perivascular lymph spaces follows with the production of the typical wheal. The white blood cells are chiefly polymorphonuclear and may contain a disproportionately large number of eosinophils. The mechanism of wheal production may be attributed to the release of H substance (histamine like substance) which then elicits the triple response (p 785). The toxic agent is not directly responsible for the wheal but must first damage the cells of the skin which thereupon liberate large amounts of H-substance. The latter in turn acts upon the blood vessels and the wheal is finally produced.

Etiology.—Urticaria is a very common condition occurring at all ages and in both sexes. It is observed most frequently in the adolescent and young adult. The chronic form is seen more often in females.

Differences of opinion exist as to the relationship of urticaria to other expressions of allergy. While it is true that there is a high incidence of positive scratch tests and passive transfer experiments the causal relation of the allergens to the eruption cannot as a rule be demonstrated. In the experience of certain observers there is an abnormally high familial occurrence of allergic diseases while others find no such hereditary predisposition in urticaria. Certain nonspecific factors contribute to an attack of urticaria by permitting the production of allergenic substances from otherwise banal foods. Ingestion of large amounts of alcohol and secretions of detoxic substances of digestion and constipation have been shown to react in this manner.

DIFFERENTIAL DIAGNOSIS OF

Wheals

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|--------------------------|---|
| Anaphylactoid purpura | Allergic dermatosis characterized by purpura and urticaria. May be associated with abdominal pains and arthralgia. Look for offending allergen or focus of infection (p. 3423) |
| Bites | Urticarial areas surrounding punctum due to bite by bedbug, flea, chigger, mite or larvae. Identify invader in local lesion (p. 3180) |
| Contact dermatitis | Local whealing from exposure of hypersensitive individuals to drugs and chemicals in cosmetics, clothing, local applications or substances handled in occupation (p. 3030) |
| Dermatitis herpetiformis | Rare skin disturbance characterized by bilateral symmetrical groups of papules, vesicles, pustules, wheals or bullae (p. 3371) |
| Drug eruptions | Diffuse urticaria due to oral or parenteral administration of therapeutic agents such as sulfonamide, barbiturates, gold, arsenic iodide, bromide, phenolphthalein, antipyrine, aminopyrine, salicylate or silver |
| Hyperthyroidism | Generalized urticaria. Particularly related to psychogenic difficulties. Note elevation of B. M. R. and therapeutic response to iodide and Deracil (p. 1197) |
| Leukemia | Urticarial dermatoses. Associated with fatal disease of blood-forming organs. Get hemogram and marrow smear (p. 1100) |
| Menstrual | Occasional urticarial formation recurring at menstrual epoch |
| Pregnancy | Occasional generalized urticaria. Particularly associated with psychogenic disturbances during course of pregnancy |
| Prurigo mitis | Atopic dermatitis of childhood characterized by fleshy papules and urticarial wheals. Seek offending allergen (p. 3343) |
| Solar dermatitis | Urticaria and herpes formation following exposure to sun |
| Physical allergy | Urticaria associated with exposure to heat, cold or water |
| Urticaria | Acute or chronic dermatosis with marked itching. May be associated with hypersensitivity to offending allergen. May be psychogenic. Note therapeutic response to epinephrine (p. 3345) |
| Urticaria pigmentosa | Rare hereditary disease with pigmented macules which wheal on physical pressure such as pinching. Note increase of mast cells in cornu. Look for offending allergen (p. 3158) |
| Vaccinia | Widespread dissemination of vaccine virus. May be associated with urticaria (p. 428) |

Erythema multiforme

Generalized dermatitis Usually involving dorsum of hands and feet and extensor surfaces of forearms and legs Usually observed in spring and autumn in patients between the ages of 10 and 30 Lesion characterized by iris colored nodules vesicles bullae and wheals (p 3374)

The specific agent usually reaches the skin from the blood stream after *ingestion* (foods drugs) *injection* (drugs biological substances) or *absorption* through accessible mucous membranes as the nose the conjunctiva the mouth the vagina and the rectum (drugs inhalants other than drugs) The noxious agent may rarely be absorbed transdermally (drugs fabrics) or it may be a *physical force* acting directly upon the skin (heat cold light trauma) The excitant is usually an exogenous substance but may be elaborated within the body in endocrine and metabolic disorders or in infectious disease *Inhalation* of certain allergenic substances is an unusual cause of urticaria Among these are *cosmetics* (orris root in face powder) *pollens* *insecticides* (pyrethrum) *animal danders* *fabrics* (silk wool) and *dyes* (hair)

Foods are the most common etiologic agents Sensitivity to foods of the *urticarial type* is not fixed A certain food may be ingested with impunity for years and suddenly give rise



Fig 984.—Urticaria

to an urticarial attack Later this food may be eaten again without trouble and eating that it is not alone the food allergen but some constitutional aggregate of metabolic or digestive disturbance which permits the production of the sensitizing allergen

Drugs are often possible for the eruption most commonly opium sulfonamides penicillin coal tar products aphenamine and its congeners tryparsamide phenolphthalein quinine cinchophen bismutates iodides and bromides It does not matter whether these drugs are introduced by ingestion injection (subcutaneous intramuscular or intravenous) or absorbed through the accessible mucous membranes (nose conjunctiva mouth vagina and rectum) Drugs applied to the broken or unbroken skin may be absorbed into the circulation and in rare instances cause urticaria

Biological products are frequent offenders especially in the characteristic syndrome known as *serum sickness* (p 548) Therapeutic serums of many kinds will excite an urticarial syndrome even after the first injection Other biological preparations derived from animal tissues such as *insulin* *liver* *stomach* *extracts* and *endocrine glandular products* may also be responsible

Focal infection of chronic low grade form may induce urticarial outbreaks Cases have been traced to infection in the teeth tonsils paranasal sinuses appendix gallbladder and the urogenital tract Rare instances have been related to chronic superficial fungus infections of the skin

An *inflammatory disease* of the skin lead to urticarial lesions in addition to the lesions characteristic of the causative agent Thus when a patient has at times seen in *scabies* and *pediculosis*

losis especially when these diseases have been present for some time. The *stings* of certain insects produce typical wheals which must not be confused with urticaria. These are seen in bites of *bees wasps mosquitoes bedbugs* and *fleas*. Their distinctive feature is the presence of a central hemorrhagic punctum where the parasite has bitten or stung.

Visceral parasitic infestations are at times attended by urticaria. These include most frequently the roundworm (*Ascaris lumbricoides*), tapeworm *echinococcus* and *schistosomes*.

Physical energies (p 3351) produce attacks in predisposed individuals. Contact with *hot or cold substances* especially water may produce the typical eruption either locally in the area of contact or less frequently in generalized form. *Exposure to light* especially certain bands of the solar spectrum may induce an attack. *Stroking or rubbing* the skin may cause a wheal in the area traumatized. This *dermographism* has been separated from true urticaria by Duke who called it *factitious urticaria*. It is not as a rule associated with pruritus as are the other forms. These varieties have been grouped together as *physical allergy* (p 3351). They are of considerable importance especially the form due to cold. Certain recent investigations have shown that shock like reactions from cold may be responsible for some cases of drowning.

Psychic influences may play a significant part in some cases. Emotional stress and sudden mental shocks have been known to precipitate the eruption. Instances have been reported in which the eruption recurred only at those times when the patient was confronted with a specific emotional situation.

Endocrine functional alterations and diseases are at times associated with urticaria. It may appear during *pregnancy* the *climacteric* and may recur during the *menstrual periods*. It is also at times met with in *hypothyroidism*, *exophthalmic goiter* and *hypopituitarism*.

Systemic diseases are occasionally accompanied by urticarial attacks not caused by the drugs used in the treatment of the disease. The eruption has been encountered in the blood dyscrasias especially chronic *lymphatic leukemia* and *Hodgkin's disease*, *latent syphilis* and some infectious diseases (*rat bite fever*, *rheumatism* and *undulant fever*).

Clinical Course—The characteristic lesion is the *wheal* which is elevated flat skin colored or pink and varies from a fraction of an inch to the size of a man's hand. There is usually an area of *erythema* about the wheal. There may be one such lesion present but more commonly there are multiple lesions in varying phases of development. The lesions appear suddenly, swell to their maximum size and then involute rapidly, all within the space of a few hours. There is no scarring or pigmentation to mark the spot after complete disappearance of the wheal. Such wheals come and go with periods of remission throughout the duration of the urticarial attack. *Itching* is generally marked and practically always present. It may vary at different hours and certainly is conditioned by the 'temperament' of the individual. Some patients complain bitterly while others suffer very little. The eruption predominates on the *trunk* but may affect any part of the skin surface and mucous membranes.

In acute urticaria which is most commonly due to a food, drug or foreign serum, the disease is self limited and may last from less than twenty-four hours to about two weeks. The chronic form may go on indefinitely, cases of more than twenty years duration being known.

Differential Diagnosis—The sudden onset of urticaria, the characteristic lesion, the *uniformity* of the lesion and the evanescence of the individual wheal serve to present an almost unmistakable picture. Differentiation is required from the urticarial type of lesion produced by *insects* especially bedbugs (p 3180) those cases in which wheals occur secondarily in the *parasitic infestation* such as scabies or pediculosis, *factitious urticaria* or *dermographism* (p 3351) and *erythema multiforme* (p 3374) which has a different distribution and little tendency to itch.

Treatment—The treatment of *acute urticaria* differs considerably from

that of the chronic form. The acute form is self limited, lasts a variable number of days and usually does not present the complex etiologic problems attendant upon the chronic form. Most cases are due to the ingestion of a food or a drug or to the injection of a foreign serum. Certain foods are more frequently responsible (fish, shellfish, fresh berries). While certain drugs are more common offenders, any drug is a potential cause; hence all medications are stopped, the diet is made simple and alcohol is forbidden. Anti-histamine agents (p. 565) such as pyribenzamine or benadryl are ordered in 50 to 100 mg. doses four times daily.

Symptomatically, therapy calls for the relief of itching. Locally, *starch* or *bran baths* may be used, following which the skin is dusted liberally with talcum powder containing 1 per cent menthol. A number of medicaments are used systemically to relieve pruritus, the most important of which is *epinephrine* (1:1000) given hypodermically at frequent intervals as needed. *Calcium gluconate* orally or by intravenous or intramuscular injection is of dubious worth. *Sedation* does help and a favorite prescription is phenobarbital 0.03 gm. and ephedrine sulfate 0.02 gm. in a capsule three or four times daily. *Opiates* should never be used in the treatment of acute or chronic urticaria.

With this regimen the average patient is made more comfortable and recovers in a period seldom longer than two weeks. In fact it must be recognized that the majority of patients recover spontaneously without any treatment. A certain number, however, continue to have urticaria for a long period of time or have recurrent attacks at intervals.

In *chronic urticaria* the first requisite is a careful history. This will aid in the elimination of drugs and physical factors (light, heat, cold) and may also suggest some highly allergenic food as a cause. The time at which the attacks occur may lead the way to the discovery of a psychic factor, a food ingested at intervals or exposure to certain allergenic inhalants (cosmetics, fabrics) or a substance used by a spouse which may be allergenic for the marital partner.

A great variety of systemic, non-specific therapeutic procedures has been utilized. These include *calcium gluconate* (orally or by injection), *histaminase* (Torantil) orally, 2 tablets q.i.d., *ascorbic acid* orally in daily dosage of 200 to 500 mg., *vitamin B complex* orally or by injection, injections of *liver extract*, injections of *histamine* in minute doses, *thyroid extract* orally, *autohemotherapy*, i.e. injections of 5 to 10 cc. of the patient's whole blood intramuscularly into the buttock, *colon irrigations*, *ephedrine sulphate* orally, *dilute hydrochloric acid* orally, *peptone* orally before meals, injections of *splenic extracts*, inorganic arsenic and iron orally. Also recommended as of specific value is *theophylline monoethanolamine* given in capsules of 0.2 gm. three times daily. These have all been relegated to limbo by the successful introduction of anti-histamine products, particularly pyribenzamine and benadryl (p. 565).

ANGIONEUROTIC EDEMA

Angioneurotic edema is a localized, transient swelling of the skin or mucous membrane, similar in nature to urticaria. Like urticaria, it is due to a transudation of serum into the tissues but it is usually localized, solitary and massive.

Etiology—Angioneurotic edema occurs in both sexes and at all ages but is most common after puberty. In some instances there is a family history of similar attacks.

In only a small number of cases can a specific etiologic agent be found. Sensitization to a drug or a food may be responsible for the attacks. Absorption of bacterial toxins from a focus of infection plays a part in some and endocrine disorders or psychic influences in others. There is considerable similarity etiologically between angioneurotic edema and chronic urticaria.

Clinical Course—The swelling appears suddenly with few or no premonitory symptoms other than itching or burning. The size of the swelling varies within wide limits and may be small as a filbert or may involve half the head and neck. It is usually firm but may pit on pressure. It is skin colored or pinkish and not inflammatory.

The sites of predilection are the lips, eyelids, hands, feet, genitals and tongue. However any location may be involved including the mucous membrane of the larynx or of the gastro intestinal tract. Certain cases of migraine (p 1506) are believed to be due to angioneurotic cerebral edema. The swelling is transient but far more enduring than that seen in the lesions of urticaria. It usually lasts from twenty four to forty eight hours.

In cases where the gastro intestinal mucosa is affected there may be colicky pain, nausea and vomiting and differentiation from a surgical abdominal condition (p 1748) is extremely difficult. The most serious type is that in which the larynx is involved since the edema may completely occlude the passage and lead to death by asphyxia.

Differential Diagnosis—The clinical syndrome is so characteristic that confusion with other conditions is rare except in cases affecting the larynx, the gastro intestinal tract or the cerebrum. The sudden advent of a non-inflammatory painless swelling and the absence of fever and systemic symptoms aid in ruling out localized infections as cellulitis or abscess.

Treatment—Every effort should be made to find a specific causal allergen. All drugs which the patient ingests deliberately or accidentally (as iodides in iodized salt) should be stopped. Scratch tests with food allergens do not generally lead to the discovery of a pertinent agent. However elimination diets (p 562) may prove of considerable value. Thorough physical examination should be made for focus of infection and evidences of endocrine dysfunction. These should be treated in accordance with their demands. Psychic conditions which can be favorably influenced should receive proper attention.

The treatment of the attack depends upon its location and severity. In the laryngeal case urgent intervention is indicated and should consist of repeated hypodermic injections of epinephrine (1:1000) until relief is obtained. If the patient is cyanotic and in great distress intravenous injections of epinephrine are indicated. In serious cases where the physician has reason to believe that epinephrine will not cope with the situation it may be best to perform immediate tracheotomy (p 3958) and administer oxygen (p 3927). Patients who have a tendency to repeated attacks of laryngeal edema should carry epinephrine and a hypodermic outfit with them at all times to be ready for any emergency.

In less serious cases the various measures enumerated in the discussion of therapy of chronic urticaria (p 3349) may be used.

PHYSICAL ALLERGY

Physical allergy has been defined by Duke as that form of allergy not caused by material agents but specifically and only by the effect of a physical agent as light cold heat scratches or by physical and mental effort. Most allergists do not consider this form of untoward reaction as a true allergy since it lacks the immunologic aspects. Its mechanism may be an increase in the permeability of the cells in response to stimuli which are harmless to the average person. There follows a release of histamine which in turn produces the abnormal reactions. A similar set of symptoms can be induced by the injection of histamine hydrochloride (p 3890).

Physical allergy may result from stroking sunlight cold or heat.

Clinical Manifestations—*Urticaria dermatographica* (*dermographism* or *factitious urticaria*) is brought on by the mechanical friction of scratching or stroking the skin. There may or may not be itching and the condition may last for many years or for a lifetime.

Urticaria solaris is an eruption of wheals appearing after exposure to sunlight or artificial ultraviolet light. The eruption generally lasts about half an hour but may persist for a longer time. When exposure to strong sunlight is prolonged the reaction may become intense and endure for longer periods. Pruritus is practically always present. This reaction occurs chiefly in the exposed areas but remote covered regions may also be affected.

Urticaria hiemalis is a most important form of untoward reaction to cold. It is usually brought on by exposure to low temperatures. Cold winds may cause coryza and cough. The drinking of ice water may bring on swelling of the lips and tongue and a spasmodic cough. Immersion of the hands in cold water produces swelling of the hands the fingers become blanched and the hands purplish red. In the skin there occur pruritus paresthesias urticaria and angioneurotic edema.

Recent investigations have shown that shocklike reactions with collapse may take place especially in the act of swimming as a result of cold sensitivity. This may explain some cases of unexplained drowning. Other systemic symptoms that appear are headache tachycardia palpitation hypotension gastric hyperacidity and the presence of cold agglutinins (p 3711) in the blood.

Urticaria ab igne is an uncommon variety wherein pruritus and urticaria develop as the result of hypersensitivity to heat.

Duke has also described remote reactions of a reflex like character which are due to sensitiveness to heat and cold or which come on after physical or mental effort. These are most common after sudden sharp temperature changes. In these instances many internal tissues may be affected and attacks of bronchial asthma (p 2101) are precipitated. In the skin there may be erythema pruritus urticaria angioneurotic edema and dermatitis.

Treatment—*Dermographic urticaria* is best treated by daily brushing of large areas of the skin with a stiff brush. This may be done each morning in bed or in a warm bath. While the whealing tendency may still remain the annoying pruritus is often completely relieved.

Solar urticaria may be helped by gradually increasing exposures to

ultraviolet light Short exposures of small areas of the skin should be used in the beginning Anti histamine agents (p 565) particularly pyribenzamine and benadryl in 50 to 100 mg four times daily, afford striking symptomatic relief

Cold urticaria is a very important variety When a shocklike reaction or collapse occurs injections of epinephrine should be used A number of successful therapeutic results have been reported by the administration of histaminase (Torantil) orally and gradual desensitization One method is to immerse the hands in ice water twice daily for one minute the first week The second week this is done for two minutes the third week for three minutes, the fourth for four minutes and so on Apparently complete cure has been recorded by this procedure

Heat urticaria should be treated by using gradually increasing degrees of heat and histaminase by mouth

In all cases where therapy is of no avail the patient must be instructed to avoid the responsible factor and to shun sudden temperature changes Where the condition is serious enough to warrant it a change of occupation or climate may be recommended

BACTERIAL ALLERGIES OF THE SKIN

The presence of an infectious lesion in the internal organs or the skin may give rise to a state of altered sensitivity of the skin and other tissues demonstrable by specific testing procedures The sequence of events is (1) the occurrence of an *infectious lesion* in which the specific excitant can be demonstrated (2) intermittent or continuous showering of these organisms into the blood whence they reach the skin and induce a state of *allergy* usually of the hypersensitive (hyperallergic) variety and (3) subsequent *redissemination* of the organisms which reach the sensitized skin where they are destroyed but not without the formation of some clinically recognizable lesion

The first phase requires the proof of the presence of a *primary focus* of infection with its viable organisms Such a focus may be of short duration and may be found in any internal organ or in the skin itself The primary focus may be active or quiescent at the time of its discovery

The second phase of *cutaneous hypersensitivity* is confirmed by the increased reactivity of the skin to contact with the specific excitant The usual material used for testing the skin is an antigen containing killed micro organisms prepared from a growth upon artificial culture media This is introduced into the skin by the intradermal or cutaneous scratch method (p 558) A positive reaction consists of the formation of an inflammatory papule after twenty four to forty eight hours sometimes longer This reaction is of the delayed type as contrasted with the immediate or urticarial type of reaction seen with food extracts and other protein materials in asthma and hay fever

The third phase in which secondary manifestations (*ids*) have appeared in the skin depends for its corroboration upon the demonstration of the first and second phases The secondary lesions usually appear more or less suddenly and a search for the offending organism is practically always crowned with failure Yet in similar cases at one time or another investigators have demonstrated the presence of the organisms in scant

numbers. The histopathology of the secondary or allergic lesion has a definite resemblance to that of the primary lesion.

As presented this is the general concept of the pathogenesis of allergic manifestations in the skin of bacterial infection. Other ideas prevail notably the belief that the lesions differ not because of the altered state of reactivity of the skin but rather because the organism has become altered. It is alleged and there is some experimental proof that a decrease in the virulence of the organism leads to its rapid demise in the skin and the production of a lesion which is atypical and sterile.

The relationship of bacterial allergy and immunity is far from settled. Some argue that increased resistance to infection runs *pari passu* with increased sensitivity whereas others insist that they are diametrically opposed. The proponents of the identity of immunity and bacterial allergy believe that the processes are the same in all respects except the quantitative one. When antibodies are present in large numbers immunity exists in small numbers a hypersensitive state is present. The opposing group believes that the two processes are independent that the hypersensitive state not only does not confer immunity but is actually harmful to the individual. They claim that immunity can exist without allergy.

Tuberculosis is the classical bacterial disease associated with allergic manifestations in the skin. The presence of cutaneous hypersensitivity to *tuberculin* (old tuberculin or purified protein derivative) is readily demonstrable in a large part of the population. The significance of the positive tuberculin test in so far as its relation to visceral disease is concerned is discussed elsewhere (p. 262). At any rate the presence of a primary pulmonary focus and cutaneous hypersensitivity is the framework for the production of allergic skin manifestations (*tuberculids*). It is impossible to explain why these eruptions occur only in certain individuals. Their condition otherwise does not appear to differ in any respect from the greater number who do not develop tuberculids.

Dermatophytosis is a disease of the skin itself in which cutaneous hypersensitivity frequently supervenes. Allergic eruptions (*dermatophytids*) of various types occur and are described elsewhere (p. 3299).

Bacterial infections due to *staphylococci*, *streptococci* and other organisms are said to induce a state of hypersensitivity of the skin which may in turn lead to the production of allergic skin manifestations. These are called *bacterids* but the proof of their identity and causation is still so nebulous that it seems best not to give them any consideration here.

Syphilis manifests altered reactivity of the skin early and late in its course. During the primary period when the chancre is present it is possible to inoculate the patient in a healthy site and produce an atypical chancre. However once the secondary eruption appears it no longer becomes possible to produce a chancre in the patient. The syphilitic gumma is explained by many as an allergic manifestation. The character of the lesion its destructiveness and the absence of spirochetes can best be explained as an intense local reaction of hypersensitive tissues to a small number of spirochetes which are promptly destroyed.

Various other infectious diseases reveal a specific altered reactivity of the skin which may be useful as diagnostic aids. These are discussed more fully in their proper place but the more important ones are the *Fries test*

ultraviolet light Short exposures of small areas of the skin should be used in the beginning Antihistamine agents (p 565) particularly pyribenzamine and benadryl in 50 to 100 mg four times daily afford striking symptomatic relief

Cold urticaria is a very important variety When a shocklike reaction or collapse occurs injections of epinephrine should be used A number of successful therapeutic results have been reported by the administration of histaminase (Torantil) orally and gradual desensitization One method is to immerse the hands in ice water twice daily for one minute the first week The second week this is done for two minutes the third week for three minutes the fourth for four minutes and so on Apparently complete cure has been recorded by this procedure

Heat urticaria should be treated by using gradually increasing degrees of heat and histaminase by mouth

In all cases where therapy is of no avail the patient must be instructed to avoid the responsible factor and to shun sudden temperature changes Where the condition is serious enough to warrant it a change of occupation or climate may be recommended

BACTERIAL ALLERGIES OF THE SKIN

The presence of an infectious lesion in the internal organs or the skin may give rise to a state of altered sensitivity of the skin and other tissues demonstrable by specific testing procedures The sequence of events is (1) the occurrence of an *infectious lesion* in which the specific excitant can be demonstrated (2) intermittent or continuous showering of these organisms into the blood whence they reach the skin and induce a state of *allergy* usually of the hypersensitive (hyperallergic) variety and (3) subsequent *redissemination* of the organisms which reach the sensitized skin where they are destroyed but not without the formation of some clinically recognizable lesion

The first phase requires the proof of the presence of a *primary focus* of infection with its viable organisms Such a focus may be of short duration and may be found in any internal organ or in the skin itself The primary focus may be active or quiescent at the time of its discovery

The second phase of *cutaneous hypersensitivity* is confirmed by the increased reactivity of the skin to contact with the specific excitant The usual material used for testing the skin is an antigen containing killed microorganisms prepared from a growth upon artificial culture media This is introduced into the skin by the intradermal or cutaneous scratch method (p 558) A positive reaction consists of the formation of an inflammatory papule after twenty four to forty eight hours sometimes longer This reaction is of the delayed type as contrasted with the immediate or urticarial type of reaction seen with food extracts and other protein materials in asthma and hay fever

The third phase in which secondary manifestations (*ids*) have appeared in the skin depends for its corroboration upon the demonstration of the first and second phases The secondary lesions usually appear more or less suddenly and a search for the offending organism is practically always crowned with failure Yet in similar cases at one time or another investigators have demonstrated the presence of the organisms in scant

numbers. The histopathology of the secondary or allergic lesion has a definite resemblance to that of the primary lesion.

As presented, this is the general concept of the pathogenesis of allergic manifestations in the skin of bacterial infection. Other ideas prevail notably the belief that the lesions differ not because of the altered state of reactivity of the skin but rather because the organism has become altered. It is alleged and there is some experimental proof that a decrease in the virulence of the organism leads to its rapid demise in the skin and the production of a lesion which is atypical and sterile.

The relationship of bacterial allergy and immunity is far from settled. Some argue that increased resistance to infection runs *pari passu* with increased sensitivity, whereas others insist that they are diametrically opposed. The proponents of the identity of immunity and bacterial allergy believe that the processes are the same in all respects except the quantitative one. When antibodies are present in large numbers, immunity exists; in small numbers, a hypersensitive state is present. The opposing group believes that the two processes are independent, that the hypersensitive state not only does not confer immunity but is actually harmful to the individual. They claim that immunity can exist without allergy.

Tuberculosis is the classical bacterial disease associated with allergic manifestations in the skin. The presence of cutaneous hypersensitivity to *tuberculin* (old tuberculin or purified protein derivative) is readily demonstrable in a large part of the population. The significance of the positive tuberculin test in so far as its relation to visceral disease is concerned is discussed elsewhere (p. 262). At any rate, the presence of a primary pulmonary focus and cutaneous hypersensitivity is the framework for the production of allergic skin manifestations (*tuberculids*). It is impossible to explain why these eruptions occur only in certain individuals. Their condition otherwise does not appear to differ in any respect from the greater number who do not develop tuberculids.

Dermatophytosis is a disease of the skin itself in which cutaneous hypersensitivity frequently supervenes. Allergic eruptions (*dermatophytids*) of various types occur and are described elsewhere (p. 3209).

Bacterial infections due to *staphylococci*, *streptococci* and other organisms are said to induce a state of hypersensitivity of the skin which may in turn lead to the production of allergic skin manifestations. These are called *bacterids* but the proof of their identity and causation is still so nebulous that it seems best not to give them any consideration here.

Syphilis manifests altered reactivity of the skin early and late in its course. During the primary period when the chancre is present, it is possible to inoculate the patient in a healthy site and produce an atypical chancre. However, once the secondary eruption appears, it no longer becomes possible to produce a chancre in the patient. The syphilitic gumma is explained by many as an allergic manifestation. The character of the lesion, its destructiveness and the absence of spirochetes can best be explained as an intense local reaction of hypersensitive tissues to a small number of spirochetes which are promptly destroyed.

Various other infectious diseases reveal a specific altered reactivity of the skin which may be useful as diagnostic aids. These are discussed more fully in their proper place, but the more important ones are the *Frei test*

(p 472) for lymphogranuloma venereum, the *Foshay test* (p 323) for tularemia the *mallein test* (p 327) for glanders the *coccidioidin test* (p 499) for infections due to *Coccidioides immitis* the *brucellergen test* (p 317) for brucellosis and the *Ducrey* or *Ito Reenstierna test* (p 289) for chancroidal infection

Certain characteristic but not specific eruptions as *erythema nodosum* and *erythema multiforme* are considered as allergic manifestations. They may occur in a variety of infectious diseases. *Erythema nodosum* (p 3377) frequently appears in the course of coccidioid bronchopneumonia and may occur as a part of rheumatic fever streptococcal pharyngitis tuberculosis syphilis lymphogranuloma venereum or dermatophytosis. In each of these conditions the dermatosis represents a nonspecific allergic manifestation of the infection.

The skin also acts as an indicator of immunity to infection as in the *Schick test* (p 304) for diphtheria and the *Dick test* (p 183) for scarlet fever. Here a negative reaction (anergy) to the specific testing material is ordinarily a sign of immunity to the disease.

CHAPTER 150

THE DESCRIPTIVE DERMATOSES

| | |
|--|--|
| Acanthosis Nigricans | Lichen Planus |
| Acne Necrotica (Var of forms) | Lupus Erythematosus (Chronic) |
| Acne Rosacea and Rhinophyma | Lupus Erythematosus (Acute Disseminated) |
| Acne Vulgaris | Macular Atrophy |
| Acneiform Dermatitis | Milium |
| Aerodermatitis Chronica Atrophica s | Panniculitis |
| A rosclerosis | Pemphigus |
| Ainhum | Pityriasis Rosea |
| Dermatitis Hemostatica | Pityriasis Rubra Pila s |
| Dermatitis Herpetiformis | Poikiloderma Atrophica s Vasculare |
| Dermatomyositis | Psoriasis |
| Erythema Multiforme Exudativum (Heb s) | Parapsoriasis |
| Erythema Nodosum | Purpura |
| Erythroplasia | Scleredema Adulorum |
| Exfoliative Dermatitis | Scleroderma |
| Granuloma Annulare | Seborrhea |
| Granuloma Fungoides (Myco s Fungoides) | Seborrheic Dermatitis |
| Leukemia Cutis | |

The descriptive dermatoses include a wide variety of cutaneous manifestations which cannot be classified at the present time. Some (*lichen planus*) seem to be strictly cutaneous phenomena while others (*acne vulgaris*, *psoriasis*) probably represent dermatological manifestations of more profound metabolic disorders. *Pemphigus* appears to be a distinct clinical entity of unknown but uniform etiology while the *purpuras dermatitis exfoliativa* and the *erythemata* are symptomatic responses that result from a diversity of causation.

The descriptive dermatoses vary also in their significance. *Pityriasis rosea* represents nothing more than a nuisance disease whereas *mycosis fungoides* is usually a fatal disturbance.

For convenience the descriptive dermatoses are described in alphabetical order above.

ACANTHOSIS NIGRICANS

Acanthosis nigricans is a rare disease whose chief clinical features are pigmentation and papillary excrescences of the skin. A *juvenile form* appears in childhood and is of unknown causation. It has been variously attributed to endocrine dysfunction especially of the suprarenal glands to disturbance of the autonomic nervous system to chronic infection and to trauma. The adult form is almost always the consequence of carcinoma usually involving the abdominal viscera. It has also been observed in instances of metastatic abdominal carcinomatosis. While the exact mechanism is not clear it has been ascribed to affection of the autonomic nervous system or of the suprarenal glands.

The Eruption—The eruption is bilaterally and symmetrically distributed as a rule. The lesions are observed in the axillae the folds of the groins

on the genitals the cheeks the breasts and in the intergluteal and submammary regions Other areas may also be affected There are diffuse patches of *pigmentation* varying from a light brown to deep black color The *skin is thickened* and more or less profusely studded with *warty papillary or vegetating growths* In the folds the action of sweat and friction may induce a moist state with odoriferous discharge In the adult variety the cutaneous abnormality is overshadowed by the underlying malignancy which eventually leads to a state of cachexia and a fatal termination

Treatment—There is no effective therapy The juvenile examples last indefinitely There have been isolated reports of improvement following the administration of thyroid or suprarenal gland extracts The adult form has a fatal outcome as a result of the uncontrolled primary cancer

ACNE VARIOLIFORMIS (ACNE NECROTICA)

Acne varioliformis is a chronic remittent disease characterized by crops of *papulopustules on the face* When these heal they leave *pitted scars*



Fig. 985.—Acanthosis nigricans of axilla (Courtesy of Dr. Stuart C. Way)*

similar to those of smallpox The disease attacks both sexes usually after the age of thirty Its cause is unknown although pyogenic cocci are constantly present in the lesions

Eruption—The eruption distributes itself characteristically along the *hair line* and on the *nose* It extends for a short distance onto the *scalp forehead* and *cheeks* The lesions may be few or many and are indolent dull red firm *papulopustules* A central *necrotic area* forms and becomes *crusted* When healing occurs there remain deep *pitted scars*

Diagnosis—Acne varioliformis lacks the comedones of *acne vulgaris* (p 3358) and always occurs at a later age In *secondary syphilis* (p 3281) of the papulopustular variety the eruption is more widely distributed and less indolent mucous membrane lesions may be present and serologic test of the blood is positive *Drug eruptions* (p 3335) are excluded by the history The *papulonecrotic tuberculid* (p 3270) rarely occurs on the face alone and does not respond to therapy

* Andrews Diseases of the Skin

Treatment—The treatment of acne varioliformis is quite satisfactory since most patients do well with the local application of 3 to 10 per cent Ointment of Ammoniated Mercury (p 3121) the proprietary Quinolol Compound Ointment (p 3123) or Penicillin Ointment.

Patients who fail to respond or who develop sensitivity may be given the undernoted prescription

| | |
|---------------------|------------|
| R. Resorcin | 0.9 |
| Precipitated Sulfur | 1.5 |
| Cold Cream | qs ad 30.0 |
| Sg. External use | |

Röntgen therapy and **ultraviolet light** are recommended but are unnecessary in the average patient.

Many systemic preparations have also been tried in the occasional refractory case. These include the oral administration of *sulfathiazole* or *sulfadiazine* injections of stock autogenous staphylococcal vaccine or of staphylococcus toroul or ambotoxoid.

ACNE ROSACEA (ROSACEA) AND RHINOPHYMA

Acne rosacea is a chronic disease limited to the face and characterized by persistent *redness telangiectasis* and *acneform* lesions. The disease occurs in both sexes perhaps more frequently in *females*. It appears in adult life usually after the age of thirty.

Etiology—While the exact etiology of rosacea is unknown there seems to be a definite relationship to disturbances of the *gastro-intestinal tract* and the *liver*. Chronic gastritis with hypochlorhydria or achlorhydria is observed in over one third of the patients. Chronic alcoholism which may induce gastritis and liver dysfunction is often a contributory factor. Chronic constipation and gynecologic disorders are said to be predisposing and aggravating conditions. The disease is made worse by external irritation as exposure to heat wind ultraviolet light and sunlight. Recent investigations suggest a causal mechanism in hypovitaminosis especially of the B complex which may be initiated by the disturbed gastro-intestinal or hepatic function.

The Eruption—Acne rosacea is localized typically on the *nose* and adjacent parts of the *cheeks* with lesser involvement of the *middle of the forehead* and the *chin*. Rarely the entire face may be affected. The onset is insidious and gradual with flushing more intense and more persistent than usual until finally the skin becomes permanently reddened. At this stage there is a *diffuse dusky erythema* with many *telangiectatic vessels*. Later indolent red *acneform papules* and *pustules* develop. In addition there may be excessive *oiliness* and mild *seborrheic dermatitis* (p 3432).

In a very small proportion of male patients there is progression to *rhinophyma*. Here the nose becomes disfigured due to a marked hypertrophy of the sebaceous glands. It has the appearance of a dull red irregularly lobulated mass with dilated follicles and extensive telangiectasias (Fig 936).

Ocular Complications—Ocular complications are observed in a varying number of patients with rosacea. Most frequent is a marginal *blepharitis* which may lead to the formation of *meibomian cysts*. Of greatest interest and importance is the so called *rosaceous keratitis* (p 1626) which produces ulcerations of the cornea opaque scars and impairment of vision.

on the genitals the cheeks the breasts and in the intergluteal and submammary regions Other areas may also be affected There are diffuse patches of *pigmentation* varying from a light brown to deep black color The *skin is thickened* and more or less profusely studded with *wartlike papillary or vegetating growths* In the folds the action of sweat and friction may induce a moist state with odoriferous discharge In the adult variety the cutaneous abnormality is overshadowed by the underlying malignancy which eventually leads to a state of cachexia and a fatal termination

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is suggested by the modification of the *mammary glands* for the secretion of milk and of the *anogenital glands* for the secretion of odoriferous substances functioning as erotogenic agencies during heat

3 The frequent occurrence of acne in more than 50 per cent of adolescent children suggests that the condition may be *physiologic* rather than *pathologic*

4 Boys are more frequently afflicted and seem to develop the disease in a more severe form than do girls

5 Girls who develop acne usually have certain masculine characteristics The pubic hair escutcheon is often of the triangular type (p 2527) there is frequently more than a considerable amount of hirsuties and the



Fig 986—A Rosacea B Rhinophyma C Acne vulgaris D Pustular acne

female sex characteristics such as the prominence of breasts and the curves of the buttocks are not conspicuous or prominent

6 The therapeutic injection of *androgen* (p 2401) which is employed in certain types of *menorrhagia* (p 2557) produces a deepening of the voice increased hairiness and typical *acne* in women at the menopause

7 Distinctly feminine women who have round faces and prominence of the breasts and buttocks rarely develop acne which is also conspicuously absent in boys of the less obviously masculine type It is to be emphasized that this discussion has no bearing on the question of sexuality but is merely applied to the endocrinological balance

8 Finally it is a common observation that acne tends to *disappear* after the teen age and is rarely seen in adults who have passed their thirtieth birthday

Diagnosis—*Acne vulgaris* (p 3359) occurs in younger persons, there are many comedones but no evidences of erythema or telangiectasia *Seborrheic dermatitis* (p 3432) is more widely distributed it is unassociated with acneform lesions or telangiectasis and shows a yellowish greasy scale *Lupus erythematosus* (p 3262) does not often attack the chin tends to a 'butterfly' distribution shows no acneform lesions and may reveal the typical dry adherent scale and atrophic changes The *rosacea like tuberculid of Leuandowsky* (p 3270) is rare occurs further back on the cheeks is seldom located on the nose and reveals tiny apple jelly nodules on diascopy

Treatment—*Systemic therapy* is directed at the avoidance of agents which intensify hyperemia of the face The diet must be bland and simple excluding fatty and rich foods spices alcohol tea and coffee External irritation from wind sun and radiant heat is shunned as completely as possible Disturbances of the stomach and liver chronic constipation female pelvic disorders and foci of infection should receive the treatment they merit With achlorhydria or hypochlorhydria it is well to administer dilute hydrochloric acid (p 1740) while patients with hyperacidity are given antacids (p 1754) Successful results have been attained in the therapy of rosaceous keratitis by the administration of riboflavin in doses of 5 to 15 mg daily The oral use of vitamin B complex and injections of liver extract have been recommended especially for the keratitis

Local treatment is fundamentally similar to that of *acne vulgaris* (p 3362) most valuable being hot wet dressings of diluted *Vlemmichx's solution* (p 3116) *resorcin and sulfur ointment* (p 3126) or lotion *Roentgen therapy* is of benefit in clearing up the acne component but does not as a rule favorably influence the rosacea The telangiectatic vessels can be treated by means of *electrolysis or scarification* (p 3792)

Rhinophyma can be improved by shaving off the hypertrophied tissues with a scalpel or the cutting current The proper normal planes of the nose must be distinguished and preserved in this procedure If hypertrophy is very mild the condition may be benefited by applications of *trichloroacetic acid* (p 3131)

ACNE VULGARIS

The common type of acne one of the most frequent causes for dermatological consultation is a chronic inflammatory disturbance of the pilosebaceous apparatus It is characterized by excessive oiliness of the skin comedones papules pustules and cystic lesions

Etiology and Pathogenesis—*Acne vulgaris* is a familial and hereditary cutaneous manifestation of a profound metabolic disorder There is much evidence to indicate that disproportionate increase of circulating androgen constitutes the fundamental causative agency The blood of males and females alike contains estrogen and androgen in varying proportions That acne results from a dislocation of the ratio between the gonadal substances on the side of an excess of androgen is suggested by the following clinical observations

1 *Acne vulgaris* usually commences before or during puberty coincidental with the increased activity of the oil glands that characterizes this period

2 The close interrelationship between the oil glands and the gonads

The importance of the recognition of the metabolic factor in the pathogenesis of acne vulgaris is related to the attitude of the therapist. He must instruct both the patient and parents that (1) *dermatological therapy is a palliative measure which cannot be regarded as curative until puberty has passed* (2) *since the metabolic abnormality is continuing the skin requires constant attention if recurrences and scarring are to be prevented*

Exciting Causes—Innumerable agencies are believed to provoke acne though the actual relationship is difficult to prove in all but a few instances

It is a common lay belief that *masturbation produces or provokes acne* but there is not the slightest reason to substantiate this idea. In fact it is highly important for the physician to emphasize the absence of any relationship since otherwise the unfortunate child has a feeling of guilt in addition to his cutaneous difficulty and is led to believe that he or she is responsible for the dermatological blemish. False teachings of this type add to the psychological hazard that is already sufficiently crippling and may produce profound personality changes in a perfectly innocent boy or girl

A dangerous lay fallacy enunciated with the fervor of the ignorant is the tenet that *intercourse will relieve acne*. Girls and boys are told glibly that the eruption will disappear as soon as they marry. This suggestion may encourage the sufferer to seek an extramarital relationship or a hasty and injudicious marriage as a therapeutic expedient

Of the provocative factors that seem to bear a proven clinical relationship to acne are (1) the excessive ingestion of *starches and sweets* particularly *chocolate* (2) the administration of *halogens* such as *iodides* or *bromides* for therapeutic purposes (3) *constipation* may have a deleterious effect but this is by no means a consistent finding

Many adolescent patients with acne are in excellent general health and no underlying etiology can be found other than careless skin hygiene

Pathology—The acne process results from a *hypersecretion of sebum* and a *hyperkeratosis of the hair follicle*. There is an accumulation of sebum and horny material in the follicle forming a comedone. The *knugo* hair is destroyed and so in time is the sebaceous gland. There is an inflammatory reaction within and about the follicle with the production of a *papule*. When pyogenic organisms invade and infect this lesion it becomes *pustular*

Eruption—The eruption is most common on the *face* but the *neck*, *shoulders*, *arms*, *chest* and *back* are not infrequently affected. The areas may be simultaneously involved. In some instances the face alone is diseased while in others the face is clear and the trunk is affected. A variable number of *comedones*, *infected comedones*, *reddish conical papules* and *pustules* are usually present as well as marked *oiliness*. In some instances there is a dominance of a certain type of lesion such as the *pustule*, *papule*, *comedone* or a deep cystic mass containing a mixture of liquid pus and cheesy sebaceous material (Fig 986)

There is a tendency to *spontaneous healing* of the lesions. If they are superficial there may be no permanent residuum but if they are deep lesions a *scar* frequently results. *Keloids* may develop after healing especially in the large indurated papular lesions on the shoulders and trunk. The scalp is often excessively *oily* and *scaly*

DIFFERENTIAL DIAGNOSIS OF

*Common Dermatoses of Childhood and Adolescence
in Addition to Those of Newborn and Infants*

| | LESION | SITE OF PREDILECTION | REMARKS |
|--|---|---|---|
| Acne vulgaris (p 3358) | Pustules comedones | Face and shoulders | Probably due to hormonal imbalance in adolescents |
| Bedbug bites (p 3187) | Papules with punctum | Exposed parts | Examine bedding |
| Contact dermatitis (p 3330) | Eczema | Exposed parts | Such as poison ivy toilettries and chemicals Corroborate history with patch test |
| Dermatosis papulosa nigra (p 3150) | Papules | Middle third of face | In colored women |
| Erythema nodosum (p 3377) | Blue nodules | Extensor surfaces of legs | Associated with hypersensitivity usually to bacterial protein |
| Erythema multiforme (p 3374) | Red macules papules nodules vesicles and bullae | Generalized and mucosal | With fever May be sensitization to drug or to bacterial protein |
| Granuloma annulare (p 3384) | Circinate papules and nodules | Fingers hands elbows and knees | Rare disease of unknown cause |
| Keratoderma palmaris et plantaris (p 3153) | Horny thickenings | Palms and soles | Frequent in Mediterranean inhabitants |
| Lichen scrofulosorum (p 3269) | Papules | Neck and trunk | Grouped lesions in tuberculosis Get biopsy |
| Miliana (p 3169) | Papules and vesicles | Generalized | Prickly heat Usually follows fever or hot spell |
| Multiple benign cystic epitheliomas (p 3208) | Yellow nodules | Face | In females Get biopsy |
| Pediculosis (p 3182) | Itching and excoriations | Scalp body | Identify louse or nits |
| Scabies (p 3180) | Itching burrows and excoriations | Interdigital wrists abdomen genitals and buttocks | Identify acarus |
| Sudamen (p 3169) | Vesicles | Trunk | Sweat rash |
| Xanthomas (p 3244) | Yellow papules | Face eyelids arms and elbows | Look for other metabolic disturbances such as diabetes mellitus and reticulo-endothelioses (p 1946) |

mutual cooperation and a sense of humor cannot be exaggerated in the management of a disturbance which may go on for many years

There are two harmful attitudes which the physician may assume to the detriment of the therapeutic program At one extreme many competent physicians have completely lost faith in the therapeutic attack upon acne They see the patient wander from pillar to post starting with one form of therapy and progressing to another type without any degree of success In the larger communities the patient travels from one specialist to his colleague and those who start with Specialist A turn next to Specialist B while the patient of Specialist B makes his way to Specialist A The practitioner cannot be too greatly blamed if he loses confidence in the therapeutic endeavor and assumes an attitude of complete defeatism This is most unfortunate since splendid results can be obtained if reliance is placed upon elbow grease rather than elegant and complicated ointments intended for miraculous use Another attitude as unfortunate as the nihilistic view is that of the enthusiast who makes rash promises of results His inevitable failure precipitates the patient into a slough of despair and discourages him from any concerted attempt to control the cutaneous affliction

The happiest results in acne are achieved by the cooperation of patient and physician Especially in the youthful patient it is wise not to make the daily therapy excessively burdensome or time consuming Once weekly or bi weekly the child should visit the practitioner who can *extract comedones and evacuate pustules* with a minimal amount of trauma

Specific Therapy—An approach to specific therapy involves the injection or administration of *estrogen* to compensate for the relative increase in androgen This procedure unfortunately is not without danger since the hormone may produce other undesirable systemic effects Girls may have excessive or irregular menses and the consequences in adolescent boys may be disturbing from many angles not the least of which are the possible alterations in personality and sexuality Serious studies now in progress may clarify the situation but for the present practitioners should reserve estrogenic therapy for unusual and resistant examples

Local Treatment of Skin—An advantageous routine for the patient consists of a thorough washing of the face with soap and warm water before bedtime Ordinary *white soap* is used unless the skin is excessively oily when *Tincture of Green Soap* is substituted Following the face wash the face is immersed in a hot solution of saturated *boric acid* or the solution is applied in the form of a compress After the face bath the skin is dried and a lotion or ointment applied according to directions

The patient is warned to *avoid squeezing blackheads* These are left for the gentler ministrations of the physician who removes the *comedones* with a *comedone extractor* devised for this purpose If a comedone does not come out easily it may be possible to loosen it by inserting the point of a fine blade into the follicle It is better to leave the comedone in situ rather than injure the tissues in a violent attempt to dislodge it Small *pustules* should be evacuated with care The cheesy plug is emptied as well as the small amount of purulent matter After dealing with a larger fluctuant cystic lesion a small dam is inserted to prevent refilling

Prescriptions for Local Use—The helpful topical applications in acne

Diagnosis—In the adolescent period the diagnosis of acne vulgaris is simple. The only eruptions that may occur at this time and may cause confusion are the acneiform lesions due to *iodides* or *bromides* and *neurotic excoriations*. The *halogen acne* (p 3340) is more acute in its onset and comedones are frequently absent. *Neurotic excoriations* (p 3231) usually occur in girls, the lesions are crusted and may be present on the extremities, comedones are absent and the patient will usually admit the etiology of the eruption.

In older persons acne lesions are produced by exposure to tars, petroleum oils and chlorine compounds. These are seen almost exclusively in males; they affect the extremities more commonly than the face and the nature of the work suggests the causation. *Acne rosacea* (p 3357) is generally confined to the middle portion of the face; erythema and telangiectases are more marked. *Acne varioliformis* (p 3356) is composed of pustular lesions situated along the hairline and on the nose; the varioliform scar is typical and comedones are absent. Infected cystic acne must be differentiated from *simple furuncles* which are more acute, superficial and responsive to therapy. Other evidences of acne are usually lacking in the case of furuncles.

Treatment—The present state of knowledge does not permit the use of a concerted therapeutic program aimed at the fundamental metabolic disturbance that causes acne vulgaris. Topical treatment constitutes the most practical and safe approach to the management of the condition and its persistence and patient application require the closest cooperation and understanding between doctor and patient. If either fails in his obligations the relationship ceases. The practitioner loses his patient and a fragment of his reputation; the patient starts on the road of medical shopping that will soon exhaust the legitimate medical channels and proceed to the twilight zone of miracles and charlatanism. The inevitable outcome is a patient whose scarring is not confined to the face and who has suffered a profound personality change and a loss of faith in doctors and medicine.

The treatment of the patient with acne requires first a lucid explanation of the problem and the intended course of treatment. The child and his parents are to be informed that this disease is self limited and not curable. Treatment is aimed at its *control* and hence must be continued through adolescence. The child particularly is to be reassured that the disturbance is not a stigma and that with a few tangible exceptions nothing in his personal hygiene and habits contributes to the affliction.

The few *prohibitions* that are strictly warranted by the facts are the avoidance of excessive amounts of starches and sweets, particularly chocolate. Iodine and bromide medications are interdicted. The child is urged to attempt to have a daily evacuation though even this requirement should not be overemphasized. Girls are warned that they will probably have an exacerbation of the eruption at the menstrual time.

An assuring method of dealing with these children is to admit correctly or not that the practitioner or a parent suffered similarly at the time of puberty. The mother who usually accompanies the child to the physician should be asked to cooperate in the hygiene of the skin later to be detailed. The child is urged to submit to the maternal ministrations without excessive reluctance or obstinacy. The importance of teamwork

gen sulfide over a longer period of time than *lotio alba*. It is stronger in action and must be diluted at first.

Formula No 5 contains as written 1 per cent resorcin and 5 per cent sulfur. If well borne these concentrations are increased gradually to 5 per cent and 10 per cent respectively. *Formula No 6* is a stronger paste containing approximately 6 per cent of sulfur and 3 per cent of resorcin.

Formulas No 7 and No 8 are potent *peeling preparations* and must be used cautiously. They are applied to the face and allowed to remain in place for ten to thirty minutes after which they are removed completely with oil or cold cream. They induce marked inflammatory reaction and desquamation. They are indicated in stubborn acne when *roentgen therapy* has failed. By careful adjustment of the strength of the active ingredients and by varying the duration of application some control of the degree of reaction may be exercised.

Peeling may be induced by the application of a slush of carbon dioxide snow and acetone to which sulfur may be added. This procedure requires considerable experience and should not be undertaken without special training. The solid carbon dioxide is mashed to a fine powder in a mortar and pure acetone added until a slushy mixture is obtained. The mixture is rubbed into the skin with a piece of cotton or gauze and this procedure may be repeated several times at each sitting. The skin becomes momentarily whitened and soon after the treatment is over it appears distinctly reddened. The following day there is mild redness and scaling. This technic has been highly recommended for resistant acne and for the improvement of acne scars though its effect upon the latter is questionable.

Local Treatment of Scalp—The presence of an oily scaling scalp has an adverse influence upon acne vulgaris and may often make the condition refractory, especially along the hairline. The scalp should be thoroughly shampooed at least once weekly and where indicated a *lotion* or *ointment* (p 3445) massaged in nightly.

For the treatment of the oily scalp which is often associated with acne the following formula is useful.

| | |
|--|--------------|
| Resorcin Monoacetate | 50 |
| Bichloride of Mercury | 0.2 |
| Spirit of Lavender | 800 |
| Alcohol (70 per cent) | q.s. ad 2400 |
| Sug. Massage into scalp nightly with the fingers or a soft toothbrush. | |

Physiotherapy—Physiotherapeutic procedures are beneficial and include ultraviolet light, cold quartz light and roentgen rays. *Ultraviolet light* (p 3794) is not curative but is a valuable adjuvant in the superficial adolescent forms. It may be of assistance when roentgen therapy has failed. *Cold quartz light* (p 3794) does not tan the skin and is indicated primarily for the production of *desquamation*. It may be used in place of the chemical peeling pastes and serves the same functions.

Roentgen Therapy—The most successful single therapeutic procedure in acne is superficial roentgen therapy. It should not be used in adolescents. Most dermatologists prefer not to apply it until the patient has reached the age of 18. This modality has been variously claimed to yield 70 to 90 per cent permanent cures with one course of treatment. However it must be handled by those expert in its administration. It may be said

are those which produce a *mild desquamation* and *drying* of the skin and contain as their principal ingredients *sulfur* (p 3128) and/or *resorcin* (p 3126) They may be prescribed as *lotions ointments* or *pastes* Representative formulas are the following

| | | |
|-----|--|--------------|
| 1 R | Lotio Alba (N.F) | |
| 2 R | Precipitated Sulfur | 60 |
| | Glycerin | 100 |
| | Alcohol | |
| | Lime Water | aa 300 |
| | Rose Water | qs ad 1200 |
| 3 R | Liq Calcis Sulfuratae (Vlemmckx's sol) diluted with hot water (1 10 to 1 40) | |
| 4 R | Sat Sol of Zinc Sulfate | 240 |
| | Rose Water | 160 |
| | Filter and add | |
| | Liq Calcis Sulfuratae | 600 |
| | Glycerin | 100 |
| 5 R | Resorcin | 05 |
| | Precipitated Sulfur | 30 |
| | Cold Cream | 600 |
| 6 R | Resorcin | 10 |
| | Precipitated Sulfur | 20 |
| | Hydrous Lanolin | 50 |
| | Zinc Oxide | |
| | Petrolat Alba | aa qs ad 300 |
| 7 R | Betanaphthol | 20 |
| | Precipitated Sulfur | 120 |
| | Green Soap | |
| | Cold Cream | aa qs ad 300 |
| 8 R | Resorcin | 150 |
| | Zinc Oxide | 40 |
| | Kaolin | 10 |
| | Benzoinated Lard | 300 |

NOTES ON THE PRESCRIPTIONS—*Lotions* are preferred when the skin is very oily and *ointments* are used for the patient with a dry skin It is best to start treatment with *weaker concentrations* of the active ingredients as lotio alba (half strength) or Formula No 2 with 5 per cent sulfur If the skin tolerates the medicament well the full strength may be used

Vlemmckx's solution as a hot wet dressing is particularly valuable in cases of indurated cystic and pustular acne

Formula No 4 has the virtue of retaining an active amount of hydro

ably younger or older age. The cause is unknown but *endocrine dysfunction* is believed responsible in many instances.

Eruption—In the beginning the affected area is diffusely *reddened edematous* and *infiltrated* resembling the early stage of *scleroderma* (p 3427). Subsequently the swelling disappears and the skin becomes *atrophic* dull red or brownish red wrinkled and paper thin. There is loss of *elasticity* and *subcutaneous fat*. Through the translucent thinned out skin the *veins* and *tendons* are clearly distinguishable. The lesion may not progress beyond this point but in some cases there develops a *scleroderma* like dense inflexible *thickening of the skin*. The latter change is most often observed about the ankles where chronic *ulcers* may appear. Subcutaneous firm fibrous *nodules* may also be present within the atrophic region. A number of examples of degeneration into *squamous cell epithelioma* have been recorded.

The *extensor surfaces* of the *upper and lower extremities* are usually involved but the process may extend onto the lower portion of the trunk. The site of predilection is the *dorsum of the foot* and the *extensor aspect of the leg up to the knee*. Usually the condition is *bilaterally symmetrical* but unilateral involvement is occasionally seen. Abortive examples are observed with only 1 or 2 small atrophic areas.

Treatment—Because of the usual onset of the disease about the time of the climacteric *estrogenic therapy* (p 2515) may be of value. Locally the use of an *oil* or *cold cream* helps to soften the skin. Once the stage of atrophy is reached treatment is ineffective. Periodic inspection is important to discover evidence of malignant change (p 3209).

ACROSCLEROSIS

Acrosclerosis is a rare disease occurring chiefly in *women* and showing characteristics which suggest *Raynaud's disease* and *scleroderma*.

Pathogenesis—The exact cause of the condition is unknown. It attacks women principally from the postpubertal years up to the menopause. Some investigators believe that acrosclerosis is a variant of *Raynaud's disease* (p 1000) in which the vasomotor symptoms precede or accompany the cutaneous manifestations. Others are inclined to group it as an unusual type of *scleroderma* (p 3427). It differs however from the progressive widespread form of *scleroderma* in many ways especially in its limited distribution and in the prognosis. *Scleroderma* progresses fairly rapidly does not have the same tendency to arrest and often leads to a fatal outcome.

Eruption—The disease usually commences in the *hands* with the manifestations of *Raynaud's syndrome* (p 1000). These symptoms are of variable duration and are followed by the typical skin changes. The *face* and *hands* are always involved while the *feet* and *toes* may rarely show a mild degree of the condition. The skin of the face is drawn and taut the face being immobile and expressionless. The lips are thin the mouth narrowed the chin tapered and the nose sharp and pinched. The neck may also be affected. At times a mild degree of *hyperpigmentation* is present.

The *fingers* become rigid in a partially flexed position and movement of the hands is markedly limited. The skin of the phalanges is very hard and cannot be lifted away. The fingers are rounded or tapering. This in

that the belief that scarring is worse after radiotherapy has been adequately disproved by clinical and experimental observation

Systemic Therapy—The female with acne is advised to discontinue the use of *cosmetic creams*. She may, however, use lipstick and face powder. Special face powders containing sulfur are available and may be helpful.

Dietary regulation need not be stringent. A *simple mixed diet* is advised. Highly indigestible foods, rich gravies, excessive fat, sweets and chocolate are avoided. *Iodides* which may be present in iodized table salt and *bromides* are tabu.

Fresh air, sunshine, regular exercise and an adequate amount of sleep are of benefit. Coexistent constitutional disturbances should be treated even though no direct relationship to the acne is evident. Improvement in general health is desirable. Secondary anemia, focal infection, hypovitaminosis, chronic constipation and weight disturbances should be rectified. In many instances the basal metabolic rate is subnormal and corrective doses of thyroid extract (p. 1189) are valuable despite the minute traces of iodide.

Many therapeutic procedures have been reported successful in isolated instances where ordinary measures have failed. These include the administration of *foreign protein*, *stock staphylococcus vaccines*, *autogenous vaccines*, *staphylococcus toxoid* and *ambotoxoid*, *inorganic arsenic* (Fowler's solution) and repeated injections of *tuberculin*. These measures are not recommended for routine use. They are desperation remedies to be employed only when the patient is discouraged and demands active treatment. Any successful issue is probably *vis mediatrix naturae* (p. 3753).

ACNEFORM DERMATOSES

The acneform dermatoses include chiefly the *halogen acnes* (p. 3340) caused by the ingestion of *iodides* and *bromides* and the *occupational acnes* due to direct exposure to mineral oils, machine oils, tar, pitch, chlorine, chlorinated naphthalenes and other substances. Uncleanliness and the wearing of clothes soaked with the oily materials predispose to the eruption. The condition is not due to hypersensitivity but to irritation of the *pilosebaceous apparatus* by *fat soluble chemicals* or contained particles of metal.

The lesions appear on the *face*, especially about the eyes, the *forearms*, the *thighs*, *legs* and at times on the *trunk* and *penis*. They consist of comedones, papules and pustules. (Fig. 986.)

Prophylactic measures in industry include proper plant *ventilation*, the wearing of an *oil impervious outer garment* and *protective devices* for the face when fumes are responsible. At the termination of the work day the entire body should be scrubbed. The treatment of the skin condition is in principle that of *acne vulgaris* (p. 3362).

ACRODERMATITIS CHRONICA ATROPHICANS (DIFFUSE IDIOPATHIC ATROPHY OF THE SKIN)

Acrodermatitis chronica atrophicans is an uncommon form of diffuse *atrophy* that affects the upper and lower extremities. The disease usually attacks *females* after the age of forty although it may begin at a consider-

| | |
|-------------------------|---|
| <i>Filiformis rosea</i> | Benign generalized dermatosis characterized by scaling macules on trunk and extremities. Accompanied by severe pruritus. Note herald patch with central area resembling cigarette paper (p 3410) |
| Psoriasis | Chronic recurrent dermatosis characterized by scaling papules on trunk, elbows, extensor surfaces of forearms, knees, leg, scalp, sacrum and nails. Accompanied by intense itching. Note freedom from eruption of face, hands and toes (p 3414) |
| Scabies | Infestation with <i>acarus</i> . Note itching burrows between fingers, on webs and volar aspects of wrist, in axillary folds, on nipples, buttocks, scrotum and penis. Isolate invader from lesion (p 3180) |
| Sebaceous cysts | Large non-inflammatory tumors containing cheesy material attached at one point to skin (p 3208). Excise |
| Seborrheal keratoses | Pigmented, scaling warts. A precancerosis. Get biopsy (p 3217) |
| Steatocystoma multiplex | Rare dermatosis with myriads of small sebaceous cysts appearing on skin (p 3207) |
| Spina bifida | Dimple of skin over sacrum with small tufts of hair. Overlying congenital failure of fusion of spinous processes of lower vertebrae (p 2820) |
| Seborrheal dermatitis | Dry or greasy scales overlying pigmented macular lesions. Usually over sternum. Associated with dry or oily seborrhea of scalp (p 3432) |
| Syphilis | Generalized macular, papular, pustular or vesicular lesions. Get darkfield microscopy in addition to serology (p 45) |
| Syringocystoma | Rare dermatosis in which multiple small cystic tumors appear over anterior surface of trunk of young females. Get biopsy (p 3209) |
| Sudamen | Multiple non-inflammatory crystalline vesicles appearing in feeble, debilitated patients (p 3169) |
| Tinea corporis | Ringworm of body with scaling macular lesions. Identify pathogen by smear or culture (p 3293) |
| Tinea versicolor | Pigmented ringworm. Identify pathogen by smear and culture (p 3300) |
| Tuberculosis | Papulonecrotic encrusted lesions appearing on extensor surfaces and trunk. Get biopsy (p 3258). Lichen scrofulosorum consisting of follicular papules on trunk or neck of children. Get biopsy (p 3269) |
| Urticaria pigmentosa | Pigmented macules which wheal when rubbed. A congenital dermatosis. Often associated with hypersensitivity to a specific allergen (p 3158) |
| Xanthoma | Yellowish papules or nodules. Often appearing on buttocks. In association with diabetes mellitus or hypercholesteremia associated with systemic reticulo-endothelioses (p 3244) |

DIFFERENTIAL DIAGNOSIS OF

*Dermatoses of the Torso, Including Chest, Abdomen
Back and Buttocks*

See also Arms and Legs p 3378 Scalp and Neck p 3254 Perineum and Genitals p 290

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|--------------------------|---|
| Acne vulgaris | Comedones and pustules of face neck and shoulders In adolescents (p 3358) |
| Angioma | Isolated red papules or nodules (p 3700) |
| Bowen's disease | Eroded papules on the trunk and extremities A malignant dermatosis of the elderly Get biopsy (p 3225) |
| Carbuncle | Multilocular taphylococcal infection Usually of nape of neck (p 3249) Start intensive treatment with antibiotics |
| Contact dermatitis | Usually due to wearing apparel as in diaper dermatitis Confirm by patch test (p 3330) |
| Decubitus ulcer | In bedridden patients Usually over sacrum. |
| Dermatitis herpetiformis | Bilateral and symmetrical groups of vesicles and bullae Associated with pigmentation, itching and burning Usually over scapula, with recurrent crops May appear during pregnancy (p 3371) |
| Dermatitis medicamentosa | Variegated drug eruptions due to therapeutic substances taken orally or by parenteral injection Check history (p 3335) |
| Dermatomycoses | Chronic granulomas with ulceration. Due to fungous infections Isolate pathogen by smear or culture (p 3292) |
| Exanthema | Systemic infections associated with generalized eruptions See erythematous and scarlatiniform rashes (p 180) maculopapular eruptions and vesicular and pustular eruptions (pp 412 422) |
| Furuncles | Staphylococcal pustules appearing usually at points of pressure (p 3248) Treat locally with sulfathiazole or penicillin |
| Keratosis follicularis | Congenital dermatosis with papules and greasy crusts Appearing in infancy and childhood (p 3153) |
| Miliaria | Vesicular and papular eruption with intense itching Secondary to elevation of atmospheric or body temperature (p 3171) |
| Moles | Pigmented or non pigmented, hairy or non hairy nevi Excise if possible |
| Molluscum contagiosum | A virus skin infection characterized by umbilicated vesicles containing cheesy material Predilection for face trunk and male genitals (p 3287) |
| Pediculosis | Itching and excoriation of body due to infestation with lice Look for nits on hair and lice or crabs in clothing |
| Pilonidal sinus | Chronic inflammatory tract leading to congenital cyst over sacrum Excise if possible |

DERMATITIS HEMOSTATICA (VARICOSE VEIN COMPLEX)

An *eczematous eruption* of the leg is fairly common in individuals suffering from *varicose veins* of long duration. It results from the congestion and swelling of the part and the impaired nutrition of the tissues. Pruritus leads to scratching followed by traumatic changes. The skin becomes reddened, thickened and excoriated. Vesicles and eroded areas develop. When persistent the leg becomes chronically enlarged and ulcerated.

The varicose veins must be treated with *injections of sclerosing solutions* and other measures. This may not be possible or at least advisable until the skin condition is improved. *Rest in bed* with elevation of the leg and the application of an *elastic bandage* when the patient must be on his feet will benefit considerably. If the skin is acutely inflamed wet dressings and liniments or lotions are applied. In the more chronic varieties ointments of *tar* as crude coal tar 1 to 5 per cent in paste of zinc oxide are used. *Röntgen therapy* is of value.

DERMATITIS HERPETIFORMIS (MULTIFORME)

Dermatitis herpetiformis (Duhring's disease) : a very uncommon skin disease characterized by a chronic remittent intensely pruritic grouped polymorphous eruption.

Etiology—The cause of the disease is unknown. It attacks the sexes about equally. It may occur at any age but is most frequent in the period from twenty to forty years. A variety of probable agents have been suggested as causative particularly sensitivity to bacteria, foods and drugs (the halogens). Toxemia and endocrine disturbances have been blamed. Some investigators ascribe the condition to an unknown filtrable virus. Emotional stress and psychic insults have often preceded the onset of the condition and many believe that in some instances the nervous system is involved.

The Eruption—The eruption is composed of several different elements. There may be an agglomeration of *erythematous patches*, *papules*, *vesicles*, *pustules*, *bullae* and *pigmentation*. A *wheel like lesion* suggestive of urticaria may also be present. The predominant and most typical lesions without which the diagnosis is problematical are *vesicles*, *bullae* and *pigmentation*. Many atypical examples are observed and these display a uniform monomorphous eruption with characteristic grouping, chronicity and intense itching. (See Fig 945.)

The eruption is generally *bilaterally symmetrical* and grouped. The grouping is a distinctive feature, the lesions being massed in circumscribed areas with the intervening skin appearing normal. In chronic cases the eruption may spread widely so that this phenomenon may not be so clear. The commonest regions affected are the *scapulae*, *sacrum*, *buttocks*, *elbows* and *knees*; but other areas may also be involved.

The eruption is *chronic* and *remittent*, the lesions appearing in crops lasting an indefinite time and being followed by a period of remission. This cycle of eruption and free interval may endure for many years and while causing much suffering there is little or no impairment of the general health. Burning, stinging and itching are invariably present, their absence being a powerful argument against the diagnosis. They are usually very intense and cause the patient a great deal of misery.

duration diminishes gradually towards the wrist and forearm the hands being most affected. Dystrophic changes occur leading to fissuring ulceration stellate scarring and atrophy of the skin and distortion of the nails.

The progress of the condition is variable in most instances being slow and attaining a maximum beyond which it does not advance. There may in time occur some partial *spontaneous retrogression* of the skin lesions. A moderate degree of *arthralgia dysphagia* and *myositis* may coexist.

Treatment—There is no specific therapy. The tissues are protected from injury and excesses of heat or cold. Emotional stress may cause an exacerbation and should be avoided. *Hyperthermia* and *physiotherapeutic procedures* which tend to improve local blood supply are said to be of value. *Iontophoresis* with *mecholyt* (p. 3874) and *cervicodorsal sympathectomy* have not proved of great benefit. Improvement has been reported



FIG. 110, 111, 112.—Ainhum showing the disease at its height but no gangrene (Courtesy of Dr. I. H. Manson Bahr, London, England).*

from the *enzyme treatment* which consists of injections of an insulin free extract of the *pancreas* (p. 1936).

AINHUM

Ainhum is a rare affliction of *Negroes* in which a *fibrous sclerotic ring* forms at the base of the *toe* or *finger* and gradually produces a *spontaneous amputation*. The disease is most prevalent in *Africa* and *Brazil* but is occasionally encountered in the continental *United States*. The small toe is most often involved but other toes and rarely the fingers have been attacked.

The cause of the affection is unknown. According to most prevalent belief it is the result of minor repeated injuries with the formation of a keloidal scar. If it is seen early the constricting band may be cut and circulation restored. In the later stages however amputation must be performed.

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Laboratory Data—The *histopathology* of dermatitis herpetiformis is fairly characteristic. The *blood count* may show a moderate eosinophilia. *Examination of the fluid* from a vesicle often reveals a very high percentage of eosinophils which histopathologists claim is nonspecific and merely significant of local necrosis. The administration of *potassium iodide by mouth* may produce an exacerbation of the eruption. A patch test with 50 per cent potassium iodide in petrolatum may be positive producing a papulovesicular or bullous reaction. These findings are used as confirmatory evidence in the doubtful case.

Diagnosis—*Herpes gestationis* (p 2648) is morphologically indistinguishable from dermatitis herpetiformis. Its sole difference is the fact that it appears during pregnancy usually clearing up with the termination of gravidity. Instances have been recorded where women have had two or three attacks during successive pregnancies but have been entirely free of eruption in the intervening periods.

When the patient presents a well developed picture of the disease with bilateral symmetrical distribution and grouping of the lesions intense burning and itching a polymorphous eruption (vesicles bullae and pigmentation) the diagnosis is fairly easy. The atypical case especially when bullae predominate and the rare mouth lesions are present may simulate *pemphigus* (p 3405). The presence of itching the chronicity and remittent tendency of the eruption the unimpaired general health and the polymorphous nature of the eruption speak against pemphigus.

Systemic Treatment—Although there is no specific therapy for dermatitis herpetiformis much can be done to alleviate suffering and even shorten the course of the disease. An adequate amount of *rest* is important. Often a change of scene will yield considerable benefit. Avoidance of mental stress the use of a well balanced diet and proper intestinal function are recommended. Foci of infection in the teeth tonsils or sinuses should be removed or corrected.

The variety of drugs recommended suggests that no one has any specific efficacy. The administration of *inorganic arsenic* (p 116) often relieves itching and improves the eruption. Generalized body irradiation with *ultraviolet light* is said to be beneficial. Other procedures which have been reported as useful are injections of *liver extract* *foreign protein (milk)* *injections* and *autohemotherapy*. *Calcium* orally or by injection has found favor with some dermatologists. Combinations of these therapeutic measures are often employed as for example arsenic (Fowler's solution) orally generalized ultraviolet light irradiation and autohemotherapy (injections of 5 to 10 cc of the patient's own blood intramuscularly in the buttock).

Recently success has been achieved in some cases by the administration of *sulfapyridine* (p 88) which has brought about prompt cessation of the itching followed by clearing up of the eruption. Unfortunately this does not occur in all cases nor is the result a permanent one. Relapses occur and these cast doubt on the effectiveness of the therapy since the disease is characterized by natural remissions and recrudescences. It would be worth while to observe the effect of the less toxic sulfonamides (sulfathiazole and sulfadiazine).

Local Treatment—Local treatment includes the use of *warm starch or permanganate baths* and topical applications. The *Calamine* nc Lo

tion containing 1 per cent *phenol* and 3 to 5 per cent *Liquor Carbonis Detergens* is valuable. Ointments containing 3 to 10 per cent of *Sulfur* and the various *Tar preparations* (p 3131) often give considerable relief.

DERMATOMYOSITIS

Dermatomyositis is a rare primary nonsuppurative *polymyositis*. It is accompanied by *cutaneous* and *systemic symptoms* and often terminates fatally. The disease has been observed from childhood to old age but is most frequent in the fourth and fifth decades. It affects the sexes about equally. The cause is unknown but most observers favor the infectious theory. A variety of organisms especially streptococci have been cultured from the skin and muscle but their significance is not clear.

Clinical Manifestations—There may be a precedent *upper respiratory infection* or the disease may commence abruptly with *muscular pains* and *weakness*.



1: 988.—Dermatomyositis. Two cases proved by autopsy (Courtesy of Dr. Rustin McIntosh, Babes Hospital).

Systemic symptoms are usually in evidence. *Mild fever*, moderate grades of *spleen* and *liver enlargement* and *arthralgias* are present at times. *Vasomotor disturbances* including *acrocyanosis* and a *Raynaud's like syndrome* have been observed.

In some instances the skin changes appear first; in others cutaneous and muscular symptoms arise together. The muscular pains may be generalized but often are confined to the extremities. *Symmetrical bilateral involvement* is the rule and the muscles of the shoulder girdle, neck and arms are most commonly and intensely affected. The muscles are painful and tender. In the later stages they become *atrophic*, sometimes producing *contractures*.

In addition to the skeletal musculature, the muscles of the pharynx, the larynx, the intercostal spaces and the diaphragm may be involved in the process and produce *dysphagia*, *hoarseness* and *dyspnea*. Other symptoms include weakness of the *facial muscles*, weakness and tremor of the *tongue* and *diplopia* due to extra ocular muscle involvement.

Cutaneous changes are almost always present. *Edema* is frequent and may be localized to the face and upper extremities or it may spread to involve the neck, chest, shoulders and rarely the entire body. The skin is diffusely or spottily erythematous, the color varying from pink to dusky red. The skin is *thickened*, somewhat sclerotic and cannot be picked up by the fingers as normally. In time a variable but usually slight amount of *atrophy* and *pigmentation* supervene. *Mouth lesions* appear especially stomatitis, erosions and small ulcerations. Patchy or diffuse *alopecia* is common and deposits of calcium in the skin have been reported.

Poikilodermatomyositis—Poikilodermatomyositis is a rare condition in which the clinical picture is that of dermatomyositis but where the skin manifestations are similar to those of *poikiloderma atrophicum vasculare*. It follows a course like that of dermatomyositis.

Laboratory Findings—Laboratory findings may include *secondary anemia*, mild grades of *leukocytosis* or *leukopenia*, *monocytosis*, *eosinophilia*, *increased sedimentation rate*, *albuminuria* and *creatinuria*. *Histologic examinations* of the skin and muscle may be important in establishing the diagnosis.

Course—The course of the disease is variable. Patients with an acute onset usually progress rapidly to severe involvement or *death*. Short or prolonged *remissions* occur. Complete recovery may take place but more commonly the disease attains a maximum and remains stationary with permanent residua in the skin and the muscles. Death comes to almost half of the afflicted within the first two years. The immediate cause is pneumonia, cardiac failure or respiratory paralysis.

Diagnosis—The differential diagnosis of dermatomyositis can present considerable difficulty. The edematous stage of *diffuse scleroderma* (p 3427) lacks the acuteness, the severe muscular pains and tenderness and is slower in progress. *Scleredema adultorum* (p 3426) is not accompanied by cutaneous changes, systemic symptoms, muscular pains or tenderness. *Trichinosis* (p 539) has a higher eosinophile count, muscle biopsy may disclose the parasite and the skin test with trichinella protein antigen may be positive. The skin lesions of acute and subacute *disseminated lupus erythematosus* (p 3399) are of different distribution, there are no muscle changes and histopathology is of definitive value in differentiation.

Treatment—Suggested treatments include *nutritional support*, high *vitamin intake*, removal of *foci of infection* and *hyperthermia*. *Inhalation of oxygen* is said to be beneficial and may be combined with hyperthermia. The use of the *sulfonamides* has not been encouraging but further study is necessary. Locally, gentle massage of the muscles and measures to improve the circulation are of value.

ERYTHEMA MULTIFORME EXUDATIVUM (HEBRA)

Exudative multiform erythema, as originally described by Hebra, is looked upon as a clinical and morphologic entity of unknown etiology. It is characterized by the typical morphology and distribution of its eruption, the tendency to seasonal appearance and recurrence and the preponderance of the condition in adolescents and young adults.

Etiology—The etiology of the Hebra type of multiform erythema is unknown. It occurs most frequently between the ages of ten and thirty years.

and affects the sexes about equally. Its incidence is highest in the *spring* and *autumn months*. Many believe that it is an allergic manifestation of hypersensitivity to foods or bacterial toxins. It has often been observed to occur after prolonged exposure to sunlight hence *photosensitivity* may play a significant part in the causation.

Many different bacterial, chemical, toxic and metabolic agents are capable of producing *polymorphous erythemas* which may simulate closely exudative multiform erythema. However, these eruptions generally lack several of the characteristics of the latter, particularly the typical distribution, the usual course and the seasonal incidence. They may occur in older persons who are rarely attacked by the Hebra type of multiform eryth-

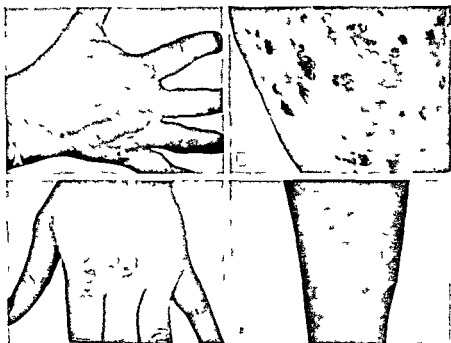


Fig. 989.—A Erythema multiforme B Erythema multiforme bullosa C Erythema multiforme nodosum D Erythema nodo um

ema. Many prefer to call them *erythema multiforme like eruptions* to distinguish them from the clinical entity under discussion. Others are inclined to group all multiform erythemas together, considering them a morphologic and pathogenic entity of multiple etiologic origin.

Clinical Manifestations—The disease may appear without prodromes or it may be preceded by a short period of malaise and slight fever. The skin lesions erupt suddenly and spread rapidly in the first few days. Ordinarily the height of the *efflorescence* is attained by the fourth or fifth day after which *involution* commences and is usually complete in ten to fourteen days. On the other hand, new lesions may appear and the disease is thereby prolonged to four weeks or more (Fig. 989).

The Lesions—The skin lesions are distributed over the dorsum of the

hands the extensor surface of the *forearms* the *dorsum* of the *feet* the extensor surface of the *legs* the *face* the *manubrium* and the back and sides of the *neck*. These are the sites of predilection but in very extensive examples they may also appear on the *palms* *soles* *arms* *thighs* *genitals* and *trunk*. In more than 25 per cent of cases notably in association with the vesiculobullous type of eruption lesions occur on the *lips* and inside the *mouth*. In a smaller percentage, *conjunctiva* and *cornea* may be involved.

Discoid Type—The usual discoid form begins as discrete red *macules* which very rapidly elevate and assume the typical appearance of round or oval sharply demarcated reddish flat *papules* or *nodules*. They vary from the size of lentils to that of lima beans. The papule enlarges peripherally the center becoming depressed and bluish while the advancing border remains elevated. Individual lesions may coalesce producing a variety of bizarre patterns. The most distinctive lesion is the *iris* figure which consists of concentric rings of different shades of color. These are produced by the recurrent outcropping of fresh papules in the center of an advancing lesion. The common discoid form of multiform erythema usually lasts about two weeks and is not associated with mucous membrane lesions.

VESICULOBULLOUS TYPE—When the exudation of fluid and cells is intense the central portion of the papule becomes vesicular or bullous (*vesiculobullous variety*). The vesicle or bulla contains a small amount of opalescent fluid and when it bursts a hemorrhagic *crust* develops.

Mucosal lesions are frequently associated. The sites of predilection are the *lips* and the *buccal mucosa* although rarely lesions may occur in the pharynx larynx and trachea. On the mucosa they begin as isolated very slightly elevated dark red inflammatory areas in which *vesicles* or *bullae* erupt. These break and lead to erosion membrane formation and rarely shallow ulcerations. When the *conjunctiva* or *cornea* is affected there is a simple conjunctivitis or keratitis which may be unilateral or bilateral. The mucosa of the male *urethra* and the female *genitals* may share in the process and lead to similar inflammatory lesions with sterile discharge. There may be mild enlargement of the *cervical lymph nodes*.

Systemic Symptoms—Systemic symptoms are usually mild and include *malaise* moderate *fever* *sore throat* and *arthralgia*. In the simple discoid variety they are often entirely absent. Involvement of the larynx and trachea may lead to mild dyspnea. The patient may complain of some burning and itching in the skin lesions and also suffer bitterly from the erosions of the lips and mouth. Severer constitutional disturbances are at times encountered in the vesiculobullous form particularly high fever and moderate degrees of prostration.

Prognosis—The prognosis is excellent for recovery although *recurrences* of the condition are frequent. Many patients have from two to five attacks. When the eruption clears it leaves no scars but a faint brown pigmentation may remain for some weeks. In the vesiculobullous variety there may be serious difficulty in the ingestion of food and fluids because of the soreness of the mouth. *Sepsis* may supervene and *bronchopneumonia* may complicate the condition. Ocular involvement particularly of the cornea may lead to scarring with impairment of *vision*. *Panophthalmitis* has also been reported.

Diagnosis—The lesion of erythema multiforme may be seen in infections of various types in metabolic disorders and after the use of drugs or the injections of sera and vaccines. It is also observed with the anaphylactoid purpuras (p 3423) to which some ascribe a close relationship. These erythema multiforme like eruptions are distinguished from the Hebra syndrome in that they are of known etiology.

Idiopathic erythema multiforme of the Hebra type generally offers no diagnostic problem. The conditions which produce erythema multiforme like eruptions must be excluded. *Acute disseminated lupus erythematosus* (p 3399) may begin with an eruption which simulates multiform erythema but the systemic symptoms and atypical skin lesions serve to distinguish the conditions. The vesiculobullous variety may closely resemble *pemphigus* (p 3405) but the occurrence of the latter in older persons and the presence of weakness, loss of weight and a positive Nikolsky sign (p 3407) characterize the more serious lesion although prolonged observation may be required before a final decision can be reached.

Treatment—Ordinarily only symptomatic treatment is required. The local application of *Calamine and Zinc Lotion* is of value. *Salicylates* (p 3832) are given if there is fever or arthralgia. The patient with an extensive vesiculobullous eruption should be put to bed. Fluids are forced and the nutrition maintained by high calory foods in liquid form if the mouth is very sore. If the patient is unable to take fluids, dextrose solution may be given intravenously.

Warm starch baths are valuable for extensive eruptions. The large bullae should be evacuated and warm wet dressings of boric acid applied. An alkaline mouth wash is used and the lips are painted with *Compound Tincture of Benzoin*. *Cold Cream* or *Boric Acid Ointment*. Recent reports claim that *nicotinamide* (100 to 200 mg doses daily) brings about a prompter retrogression of the disease. If ocular lesions are present an ophthalmologist should be summoned.

There seems to us to be no indication for the administration of the *sulfonamides* in the average case of multiform erythema. Perhaps in the very unusual instance where a patient with the vesiculobullous variety shows considerable toxicity these drugs have a place. It has been reported that *anti histamines* (p 565) have been helpful in such instances.

ERYTHEMA NODOSUM

Erythema nodosum is a morphologic entity which may coexist in the same patient with erythema multiforme. An identical eruption may be produced by diverse known agents under which circumstance the condition is termed erythema nodosum like.

Etiology and Pathogenesis—Erythema nodosum may occur at any time from childhood to old age but is most common between the tenth and fortieth year. The idiopathic variety is observed more frequently in females in whom it is commonly associated with *rheumatoid pains* and often follows a *pharyngitis* or *tonsillitis*. This has led to the belief that *streptococci* are probably responsible. The mechanism of the process is probably *allergic* since microbes are rarely found in the nodules.

In children and young adults the *tubercle bacillus* is a very common etiologic agent and there is marked *cutaneous hypersensitivity* to tuber

DIFFERENTIAL DIAGNOSIS OF

Dermatoses Commonly Encountered on the Arms and Legs

See also Hands and Feet Fingers and Toes p 3296 Torso p 3368

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|-------------------------------------|---|
| Acrodermatitis chronica atrophicans | Diffuse atrophy of skin of upper and lower extremities |
| Anthrax | Malignant pustule Note surrounding halo of vesicles Identify gram positive sporulant bacillus by smears and cultures (p 232) Start intensive treatment with penicillin. |
| Bites | Particularly those due to scabies On extensor surfaces of wrists Look for invading organisms in burrows Search clothing and bedding for pediculi (p 3180) |
| Bowen's disease | Papulo-ulcerative lesions Usually seen on trunk and extremities A malignancy of the elderly Get biopsy (p 3225) |
| Contact dermatitis | Erythematous eruption with vesiculation burning and itching From hypersensitivity to externally applied substances Poison ivy Verify by patch test (p 3330) |
| Dermatitis hemostatica | Varicose vein complex with ulceration of leg (p 3371) |
| Dermatomycosis | Deep fungous infection producing indolent granulomas with ulceration Identify pathogen by smear and culture (p 3292) |
| Dracontiasis | Filarial infection Usually noted in Nile Valley Ulcerations of legs Adult worms extracted and identified (p 3328) |
| Edema neonatorum | Swelling of lower extremities in newborn. Probably related to vitamin deficiency Note therapeutic response to high vitamin feedings (p 3151) |
| Erythema multiforme | Toxic dermatosis of dorsum of hands and feet and extensor surfaces of forearms and legs Usually seen in spring and autumn between age of 10 and 30 Note constitutional symptoms and appearance of iris colored nodules vesicles bullae and mucosal lesions Seek offending allergen (p 3374) |
| Erythema nodosum | Tender red and blue nodules of extensor surface of legs Constitutional symptoms include abdominal discomfort arthralgia and carditis Seek offending allergen often of bacterial origin (p 3377) |
| Feigned eruptions | Excoriations of arms and legs in areas accessible to scratching or the application of chemicals Get history Consider psychiatric consultation |
| Filariasis | Tropical disorder characterized by elephantiasis of legs arms scrotum or vulva Attempt to demonstrate microfilaria in blood smears taken at night (p 3321) |

| | |
|---|---|
| Gout | White nodules (tophi) usually seen in earlobes or on fingers or elbows. Demonstrate sodium urate crystals. Get blood uric acid. Note therapeutic response to colchicine (p 2867) |
| Helminthiasis | Nematode skin invasions such as dracunculiasis, filariasis, loa loa, onchocerciasis and dracuncululus |
| Idiopathic multiple hemorrhagic sarcoma | Kaposi disease. A malignant dermatosis of elderly males. Note purplish nodules on foot and ankles. Get biopsy (p 3226) |
| Lichen planus | Violaceous papules with central umbilication producing intense itching. On flexor surfaces of wrists, forearms and legs. Get biopsy. Consider psychiatric consultation (p 3389) |
| Leishmaniasis | Indolent ulcers of face or exposed surfaces of the body. Demonstrate Leishman Donovan bodies by direct smear (p 3317) |
| Lymphedema | Milroy's disease. A congenital dermatosis of puberty. With swelling of legs and no other associated abnormalities (p 3157) |
| Meralgia paresthetica | A neuritis of the anterior crural nerve with pain along anterior portion of thigh. Probably due to pressure (p 3229) |
| Necrobiosis lipoidica diabetorum | Nodular yellow infiltrations of the anterior aspect of the legs due to loss of fat. Probably the result of hypersensitivity to insulin (p 3240) |
| Neurotic excoriation | Scratch marks of forearms and other areas accessible to trauma. Consider consultation with psychiatrist (p 3231) |
| Neurodermatitis | Excoriated papules of neck and extremities with lichenification. Intense pruritus. In elderly females (p 3343) |
| Poikiloderma atrophicum vasculare | Papules with pigmentation, telangiectases and atrophy of the skin. Usual involvement of flexural regions. Get biopsy (p 3413) |
| Prurigo mitis | An atopic dermatitis of childhood. Lesions consist of fleshy papules and urticarial wheals. Seek offending allergen (p 3343) |
| Psoriasis | Chronic and recurrent dermatosis with scaling papules. Most often seen on trunk, elbows, extensor surfaces of forearm, knees, legs, scalp and sacrum. Intense itching. Note that face, hands and toes are spared but nails become involved (p 3414) |
| Scabies | Infestation by acarus. Note burrows between fingers, on web of hand, on volar aspect of wrist, in axillary folds, on nipples, buttocks, scrotum and penis. Identify parasite (p 3180) |
| Scurvy | Superficial and deep hemorrhages. Usually observed over shin. Note bleeding gums and therapeutic response to ascorbic acid (p 3238) |
| Syphilis | Copper colored macules. Often seen on lateral aspect of flanks. Psoriasis-like eruption in tertiary syphilis. Supplement serologic test with darkfield microscopy (p 45) |

| | |
|------------------------------|--|
| Tuberculosis | Papulonecrotic tuberculids on extensor surfaces in young children. Note crusting ulceration and scar formation. Get biopsy (p 3258). Erythema induratum of females. Usually seen on calves as necrotizing inflammatory nodules. Get biopsy (p 3271). |
| Xanthoma | Yellow papules and nodules. Often seen on elbows and buttocks. Associated with hyperlipemia, hypercholesterolemia, diabetes mellitus and systemic reticulo-endothelioses (p 3244). Get blood chemistry. Conduct complete physical examination. |
| Xeroderma pigmentosum | Abnormal photosensitivity of exposed parts. Note pigmented macules, areas of atrophy, telangiectases and keratoses. A precancerous. Get biopsy (p 3158). |
| Urticaria pigmentosa | Rare dermatosis in which pigmented macules wheal when rubbed. Look for presence of offending allergen (p 3158). |
| Purpura | Black and blue spots of legs and feet in young children. Note associated constitutional symptoms. Seek offending allergen, usually bacterial (p 3423). |
| Rocky Mountain spotted fever | Rickettsial infections with constitutional symptoms and a generalized petechial eruption first noted on ankles and wrists. Get Weil-Felix reaction (p 376). |

culm. Many pediatricians consider tuberculosis the only cause in children and warn that the cutaneous manifestation is a harbinger of pulmonary tuberculosis. At any rate this possibility must always be remembered and the presence or later development of active pulmonary tuberculosis excluded.

Certain drugs, especially *bromides*, *iodides*, *sulfathiazole* and the *salicylates* may elicit the eruption. Attacks have followed the injection of *diphtheria antitoxin*, *Frei antigen* and other *vaccines* and *sera*. *Coccidioides bronchopneumonia*, the so-called San Joaquin Valley Fever of California, is often associated with an eruption of erythema nodosum. It has been observed not infrequently in *lymphogranuloma venereum*, *trichophytosis* and rarely in the secondary stage of *syphilis*.

Clinical Manifestations.—The idiopathic eruption may be preceded for several days by malaise, arthralgia, mild fever and sore throat. Again these symptoms may appear simultaneously with the eruption or they may be extremely vague or entirely absent (Fig 989).

The *skin lesions* appear rather suddenly and are distributed typically on the *extensor surface* of the legs. At times there is more extensive involvement with lesions on the dorsum of the feet, the calves, the extensor surface of the forearm and rarely on the face and chest. Widespread eruptions are uncommon in the idiopathic form but are not unusual when a drug is the cause, particularly *sulfathiazole* (p 88).

The individual lesion is a firm, tender, red to bluish red, slightly elevated *nodule*. It is roughly rounded or oval and merges gradually with the surrounding normal skin. Its appearance is strongly suggestive of a

hematoma There may be only four or five such nodules but fifty or more have been observed. The nodules remain discrete and show no tendency to enlarge peripherally or to coalesce. While the lesions at times may feel soft centrally they never suppurate, ulcerate or break down. Crops of new nodules may continue to appear after the initial outbreak.

Course—The eruption generally lasts between *two and three weeks* and as the nodules are absorbed they leave behind a yellowish or greenish discoloration which resembles an ecchymosis. These pigmentations disappear in a short period of time. Unlike erythema multiforme recurrences are quite infrequent.

Diagnosis—The bilateral symmetrical distribution of the typical nodules, the abrupt appearance of the lesions, their acute course and rapid involution present a syndrome difficult to confuse with other dermatoses. *Erythema induratum* (p. 3271) usually affects the calves, is chronic and breaks down producing indolent ulcerations.

Most important in every case of erythema nodosum is the necessity for determining if possible the underlying cause. Especially in younger persons it is necessary to rule out the presence or imminence of *pulmonary tuberculosis*. *Syphilis* can be excluded by the negative serologic test of the blood.

Treatment—The disorder is self limited and treatment is symptomatic. *Rest in bed*, the administration of *salicylates* and local applications of *warm compresses* are usually adequate. Treatment with the *sulfonamides* has been recommended as a means of shortening the attack. Chronic foci of infection, especially in the tonsils, may be causative of the recurrent type and *removal of the diseased tissues* may result in permanent cure.

ERYTHROPLASIA

Erythroplasia (Queyrat) is a rare disease affecting principally the *glans penis* but also at times the *vulva*, the *lips*, the *buccal mucosa* and the *glabrous skin*. Most of the reported cases have occurred in men between the ages of thirty and fifty years. No definite etiologic agent is known.

Eruption—At the outset there appear one or more irregular, dark red patches whose surface is smooth and shiny. They are not perceptibly elevated nor is there any deep infiltration. An exudate of yellowish serum is present but no crust forms. The individual plaques enlarge peripherally and may coalesce. After a variable period of time the plaques become infiltrated, raised tumors having the unequivocal histopathology of *squamous cell carcinoma* (p. 3223). This change apparently occurs in every patient left untreated.

Diagnosis—In the precancerous phase differential diagnosis must be made from a *drug eruption*, *dermatitis venenata*, *psoriasis*, *fungus infection*, *balanitis* and *syphilis*. Careful history, complete physical examination, direct microscopic examination and culture for fungi, serologic tests of the blood, darkfield studies and observation over a reasonable interval may be necessary. In the final analysis a biopsy is essential to be assured of the correctness of the clinical diagnosis.

Treatment—The most successful therapeutic procedure has been *excision* with the scalpel or the cutting current. Removal of the local lesion

DIFFERENTIAL DIAGNOSIS OF

Scaling Dermatoses

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|--------------------------|---|
| Acrodynia | Grave systemic disturbance of infancy <i>Swelling redness and scaling particularly of hands and feet</i> Note therapeutic response to vitamin B complex (p 3145) |
| Actinic dermatitis | Following prolonged exposure to ultraviolet rays |
| Arboflavinosis | Vitamin B deficiency Associated with the angles of lips and nasolabial folds Note response to vitamin B complex (p 3238) |
| Atopic dermatitis | Infantile eczema Usually due to hypersensitivity to digestant (milk eggs p 3342) |
| Chemical exposure | As result of contact with chemicals medicinally accidentally or in industry |
| Exfoliative dermatitis | Generalized scaling of body A serious lesion often due to idiosyncrasy to treatments with arsenic gold or sulfonamides (p 3383) |
| Granuloma fungoides | Fatal systemic disorder Generalized itching scaling nodule formation tumefaction and ulceration Get biopsy (p 3386) |
| Ichthyosis | Congenital disturbance with appearance of fish scaling (p 3152) |
| Impetigo contagiosa | Staphylococcal infection of face in infancy and childhood Note pustulation and crusting (p 3251) Treat locally with sulfathiazole or penicillin |
| Keratosis follicularis | Papule formation with greasy crust. Noted in infancy and childhood Note therapeutic response to vitamin A (p 3153) |
| Keratosis blennorrhagica | Crusting and pustule formation Associated with gonococcal infection (p 3257) Treat with penicillin |
| Lupus erythematosus | Dermatosis involving middle third of face Papule formation with scaling atrophy and telangiectases Butterfly formation over bridge of nose with constitutional symptoms involving heart valves and kidneys (p 3395) |
| Measles | Systemic exanthem with branny desquamation in convalescence (p 409) |
| Neurodermatitis | Itching with lichenification Often involving neck and flexural surfaces Papule formation with scaling (p 3229) |
| Paget's disease | Of areola of breast or nipples A localized dermatitis associated with malignancy of the mammary ducts Get biopsy Prepare for radical mastectomy (p 2581) |
| Paraporiasis | A rare dermatosis characterized by papules, plaques vesicles and scaling (p 3421) |
| Pellagra | Vitamin B deficiency causing pigmentation of exposed surfaces edema redness and desquamation Associated with glossitis and peripheral neuritis Note therapeutic response to niacin (p 3239) |

| | |
|--------------------------|--|
| Pemphigus | Fatal dermatosis. Characterized by itching bulla formation of skin and mucous membranes and desquamation. Get biopsy (p 3405) |
| Pityriasis rosea | Benign generalized dermatosis with macule formation scaling and itching. Predilection for trunk and extremities (p 3410) |
| Pityriasis rubra pilaris | Papule formation around hair follicles. Black core papules of dorsum of hand with nutmeg feel. Note therapeutic response to vitamin A (p 3412) |
| Psoriasis | Scaling papules of trunk elbows extensor surfaces scalp nails and sacrum. Marked itching with chronicity and tendency to recurrence (p 3414) |
| Radiodermatitis | Scaling following exposure to x ray or radium (p 3177) |
| Seborrheal keratosis | Dry or greasy scales involving forehead, cheeks chest or the area behind the ears (p 3217) |
| Scarlet fever | Acute exanthem with erythematous eruption followed by scaling in plaques. Note Schult-Charlton blanching reaction (p 171) |
| Senile keratosis | Precancerous rough scaling lesions of exposed parts. Get biopsy (p 3216) |
| Senile atrophy | Generalized dryness of the skin. Often with itching and scaling. Try androgen or estrogen |
| Solar dermatitis | Following exposure to sunlight |
| Syphilis | Particularly palmar and plantar lesions of secondary and neonatal infection. Note positive Wassermann and therapeutic response to penicillin (p 106). Also pemphigiform syphilitic dermatitis of arms and upper trunk. |
| Tinea | Superficial fungous infections involving body scalp groin or toes. Identify fungus in scrapings |

may be adequate but in a good many instances amputation of the penis has been necessary because of recurrence

EXFOLIATIVE DERMATITIS (ERYTHRODERMA)

Exfoliative dermatitis is a generalized reddened scaling condition of the skin. It is probably never a disease *suu generis* but rather an evidence of some severe and extensive damage to the skin from *exogenous* or *endogenous* noxious agents.

Clinical Manifestations—Dermatitis exfoliativa occurs in primary and secondary or symptomatic forms. The primary disturbances which are probably very infrequent include the variety described by Wilson Brocq, pityriasis rubra of Hebra, dermatitis exfoliativa neonatorum and desquamative erythroderma. The last two are exceedingly rare and are observed only in infancy.

Exfoliative Dermatitis (Wilson Brocq)—Exfoliative dermatitis (Wilson Brocq) has been described as a primary variety of unknown etiology

It is interesting to note that this disease has almost completely disappeared from modern dermatologic literature. The most likely explanation is that instances of exfoliative dermatitis which might once have been so classified are now placed in a category of known etiology.

Pityriasis Rubra (Hebra)—*Pityriasis rubra* (Hebra) is another form of exfoliative dermatitis of unknown causation which is so rare that dermatologists with three decades of experience have never seen an example. It was once ascribed to tuberculosis although the skin did not reveal the histopathology of that disease.

Symptomatic or Secondary Dermatitis Exfoliativa—*Dermatitis exfoliativa* is usually if not invariably a secondary phenomenon occurring in the course of certain common dermatoses as the result of *drug hypersensitivity* or in association with *systemic disease*.

The dermatoses which may result in exfoliative dermatitis are *psoriasis* (p 3414) *contact dermatitis* (eczema) (p 3330) *seborrheic dermatitis* (p 3432) *lichen planus* (p 3389) *pemphigus foliaceus* (p 3405) and *pityriasis rubra pilaris* (p 3412). The transition from the localized form of eruption to the generalized exfoliative condition may be spontaneous or it may be the result of the application of strong topical remedies such as *chrysarobin*, *mercury sulfur* or *resorcin*.

Dermatitis exfoliativa may also follow contact with a volatile substance such as dry cleaning fluid in the clothes, naphthalene (moth preventive) a volatile germicide or an insecticide (Flit etc.). *Hypersensitivity to drugs* may also produce an exfoliative dermatitis. The commonest offenders are *arsphenamine*, *gold salts*, *sulfonamide* and the *barbiturates*.

Finally exfoliative dermatitis may be a manifestation of *systemic disease*, notably the *leukemias* (p 3387) and *granuloma fungoides* (p 3386).

The Eruption—The clinical picture of *dermatitis exfoliativa* is more or less the same no matter what the cause.

The skin is generally *reddened*, more or less *thickened* and *scaly*. *Alopecia* of the scalp, partial in distribution, is an almost constant concomitant. The *nails* become lustreless, discolored and deformed and may be shed although they regrow later. If the condition persists for some time the skin thickening becomes marked and there may be coincidental enlargement of the *superficial lymph nodes*. *Itching* is practically always present and may be intolerably severe. There may be vesiculation in the cases due to drugs and in contact dermatitis. However the vesicles are present only at the outset after which they are succeeded by the characteristic dry scaly eruption.

Treatment—The treatment of exfoliative dermatitis is that of the causative condition (qv). When there is no history of a precedent common dermatosis or the application of an irritating substance to the skin or of the administration of a drug, etiologic diagnosis may present difficulty. It may be necessary to make repeated studies of the blood cells, biopsies of the skin and lymph nodes and even bone marrow studies in order to reach a definite conclusion.

GRANULOMA ANNULARE

Granuloma annulare is a rare disease whose sole apparent manifestation is the *cutaneous lesion*. The condition may occur at any age but is

most frequent in *childhood* and *young adulthood*. Both sexes are affected. No etiologic agent is known but various toxic and bacterial agents have been held responsible. Many believe the condition to be related to tuberculous infection and that it is in the nature of a *tuberculid*. It is at times observed in association with active and quiescent visceral tuberculosis. The tuberculin test is more often negative than positive.

The *histopathology* is fairly characteristic revealing areas of *necrobiosis* of the collagenous tissue of the cutis about which are *epithelioid cells*, an infiltrate of *round cells* and occasional *giant cells*.

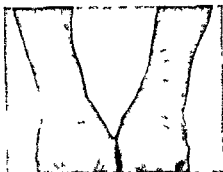


Fig 990—Exfoliative dermatitis



Fig 991—Granuloma annulare



Figs 992 and 993—Granuloma annulare (mycosis fungoides)

The Eruption—The eruption consists of skin colored or reddish firm shiny *papules* or *nodules* arranged in *circinate fashion*. The central portion of the lesion is composed of normal or somewhat reddish skin. The papular or nodular border is slightly elevated and forms a solid wavy line or is broken up into distinct closely crowded *papules* or *nodules*. Only a single lesion may be present or there may be a great many disseminated over the trunk and extremities. The individual lesion varies from 1 to 6 cm or more in diameter. The site of predilection is the dorsum of the *fingers* and *hands*, the *elbows* and *knees* but lesions may appear in any location on the extremities and trunk.

The disorder is chronic with little tendency to spontaneous involution.

The lesions tend to spread peripherally and multiply unless therapy is instituted

Treatment—The most effective procedure is *roentgen therapy* (p 3796) Many observations have been recorded in which simple *biopsy* excision, a small fraction of the lesion has been followed by complete disappearance of the entire lesion *Inorganic arsenic* (Fowler's solution p 125) is said to be beneficial Freezing the lesion with *carbon dioxide snow* (p 3785) is a satisfactory procedure

GRANULOMA FUNGOIDES (MYCOSIS FUNGOIDES)

Granuloma fungoides is a *fatal disease* of unknown etiology which begins as a *benign inflammatory process* and terminates in a *malignant neoplastic state* The condition occurs in both sexes but is more common in men It starts as a rule after the age of forty although it may appear considerably earlier or later The specific cause is unknown the disease being considered variously as a neoplasm an infection or the result of some toxin

Clinical Manifestations—It is customary to look upon the disease as a sequence through three distinct phases the *pre-fungoid* or *premycotic* the *infiltrative* and the *fungoid* or *tumorous* Occasionally lesions of the several stages coexist and in rare instances the first and second stages are completely absent The latter form is called *granuloma* or *mycosis d'emblee* (at the first onset) where the fungoid stage appears abruptly without the preliminary inflammatory or infiltrative lesions

Premycotic Stage—The premycotic stage may present any one of a number of different cutaneous manifestations none of which is characteristic clinically or histologically There may be merely intense pruritus or lesions suggestive of an *eczematous condition* of *parapsoriasis en plaques* of *psoriasis* or rarely of *exfoliative dermatitis* The eruptions are reddened scaling patches of generalized distribution which *itch* intensely although few or no scratch marks may be noted

The inflammatory lesions may clear up and recur intermittently or remain persistently until they develop evidences of infiltration This stage may last for a few months or may extend for many years In this period the diagnosis is extremely difficult often intuitive rather than factual If the histology is not specific the patient must be treated (p 577) and observed until a definitive opinion can be rendered

Infiltrative Stage—The infiltrative stage reveals dull red or brownish moderately elevated *nodules* and *plaques* These lesions are firm of various sizes and tend to form odd and unusual patterns circles segments of circles festoons and gyrate figures The infiltrative stage is of comparatively short duration tumors gradually appearing after a period of months The diagnosis is readily made from the almost unique clinical picture and confirmed by the typical histology

Fungoid Stage—In the final phase *tumors* develop in the infiltrated lesions and may be few or many in number They may be as small as a pea or large as a melon They appear as circumscribed firm smooth growths with a reddened sometimes scaly surface In time these tumors ulcerate and give rise to ugly fungating masses Diagnosis is made by the clinical appearance and histology

Visceral Manifestations—Visceral involvement may be present. Most common are regional or generalized *lymphadenopathy*, *splenomegaly* and specific tumors in the internal viscera. Death results from *cachexia* or *septicemia* in infection entering through a fungating tumor. A fatal outcome may occur rather rapidly in less than a year but some patients may survive for years with the benefit of *radiotherapy*.

Treatment—The only therapy of value is the application of *roentgen rays*. It benefits the patient in any phase of the disease and patients have been known to live more than five years receiving radiotherapy at intervals. Not all respond and in many instances response is good at first but recurrences become more and more refractory. *Inorganic arsenic* (Fowler's solution) is claimed to be a helpful adjuvant.

LEUKEMIA CUTIS

The leukemias may be associated with nonspecific cutaneous manifestations (*leukemids*) or more rarely with specific *leukemic infiltrations* of the skin.

Leukemids—The leukemids are of many varieties and only to be differentiated from similar banal lesions by careful study of the patient including examinations of the formed elements of the blood and the bone marrow where necessary.

Hemorrhages into the mucous membranes and skin and *ulcerative necrotic lesions of the gums, cheeks and throat* are seen in the acute leukemias (lymphatic and myelogenous). These may be first evidence of the disease and may be mistaken for Vincent's infection, sepsis, toxic drug reactions or purpura hemorrhagica. *Urticaria* and *erythema multiforme* are most commonly encountered in chronic lymphatic leukemia. They are transitory and evanescent but may recur and blood studies must be made to establish the diagnosis.

Pruritus is not common and may be present with pigmentation and lichenification especially in chronic lymphatic leukemia. The latter may be associated with a grouped papulovesicular pruritic eruption resembling dermatitis herpetiformis or papular urticaria.

Exfoliative dermatitis usually very pruritic may be the first manifestation of chronic lymphatic leukemia. Rare cases have been seen in which the blood showed no abnormality at first and the disease was apparently confined pro tempore to the skin. *Herpes zoster* especially when generalized and accompanied by gangrenous lesions should always rouse suspicion of chronic lymphatic leukemia.

Leukemic infiltrations—Specific leukemic infiltrations of the skin may be circumscribed and nodular or diffuse. These lesions are most commonly associated with chronic lymphatic leukemia, seldom with myelogenous or monocytic leukemia. The *nodules* are of different sizes up to a small plum and choose the face as the site of predilection but are not rare on the extremities and trunk. They are firm, reddish brownish or bluish red, attached to the skin, nonpruritic usually and rarely become ulcerative or necrotic.

The *erythrodermic form* is clinically indistinguishable from other forms of exfoliative dermatitis. Histology and blood studies are needed for differentiation. The skin represents a universal reddened and thickened ap-

DIFFERENTIAL DIAGNOSIS OF

Enanthems Involving Oral and Buccal Surfaces

Eruptions involving oral and buccal mucous membranes may represent local processes or manifestations of profound systemic disturbances. Recognition of the general character of the individual lesion may enable dentist or practitioner to arrive at a correct diagnosis.

DIAGNOSTIC FEATURES

| | |
|------------------------|---|
| Erythema | Infectious stomatitis without other manifestations. Scarlet fever with exanthem and Schott-Charlton blanching reaction. Secondary syphilis with positive darkfield findings and serology. |
| Vesicles and Bullae | Infectious stomatitis, aphthous stomatitis, herpes simplex and herpes zoster without manifestations elsewhere. Contact dermatitis from cosmetics or dentifrices. Dermatitis medicamentosa from drugs. Erythema multiforme, pemphigus and dermatitis herpetiformis with cutaneous dermatoses. Varicella and varicella with fever and exanthem. Foot and mouth disease with extremity lesions. |
| Macules and Papule | Fordyce's condition and leukoplakia without manifestations elsewhere. Lichen planus with exanthem. Measles with Koplik spots and exanthem. |
| Tumor | Epulis in pregnancy. Oropharyngeal cysts with x-ray changes and fluid by diagnostic puncture (p. 1714). Oropharyngeal neoplasms clarified by biopsy (p. 1656). Gummas with positive serology and therapeutic response to iodide. |
| Petechiae and Purpuras | Scurvy with therapeutic response to ascorbic acid. Vitamin K deficiency with therapeutic response to menadione. Leukemias and thrombocytopenic purpura with pathognomonic hemograms (p. 3704). Subacute bacterial endocarditis with positive blood culture. |
| Telangiectasi | Hereditary telangiectasis with similar lesions elsewhere and normal hemogram. |
| Ulcers | Aphthous stomatitis without lesions elsewhere. Scurvy with therapeutic response to ascorbic acid. Vitamin B deficiencies with therapeutic response to thiamine, riboflavin or niacin. Erythema multiforme, lupus erythematosus and dermatitis herpetiformis with cutaneous exanthem. Ulceromembranous stomatitis or gingivitis with Vincent organisms demonstrable on smears (p. 50). Epithelioma or carcinoma with positive biopsy. Tuberculosis with acid fast organisms demonstrable in exudate or tissue (p. 50). Diphtheria with membranous exudate and characteristic organisms in smears and cultures (p. 302). Primary or secondary syphilis with positive darkfield and/or serology. Tertiary syphilis with therapeutic response to iodide. Agranulocytosis and leukemia with characteristic hemogram (p. 3704). Typhoid fever with positive blood culture or elevated agglutinin titer (p. 60). |

Pigmentations

Normal deposits in negroes and brunettes
 Nicotine staining in habitual smokers Deposits
 of lead and bismuth with positive urine findings
 (p 3659) Adrenal cortical deficiency with hy-
 potension and asthenia

pearance with scaliness A condition formerly known as *ptyriasis rubra of Hebra* (p 3384) recognized as a fatal form of exfoliative dermatitis of unknown etiology is now credited by many with being a type of leukemia cutis wherein the blood picture may be normal or undistinctive

Diagnosis—Differential diagnosis requires studies of blood smears (p 1035) and bone marrow (p 1035) and biopsy of skin and lymph nodes

Treatment—Roentgen therapy (p 3796) is often of palliative value for the skin lesions

LICHEN PLANUS

Lichen planus is a common disease characterized by an eruption of multiple polygonal flat violaceous papules often attended by severe pruritus and lesions of the buccal mucosa The condition occurs chiefly in adult life between the ages of twenty and fifty years Instances in younger and older individuals are not rare The sexes are about equally affected

Etiology—The etiology of lichen planus is unknown Attempts to find bacteria in the lesions and in the blood stream have been attended by failure or inconsistent results Efforts to culture a filtrable virus have given negative results Focal infection and vitamin deficiency have been suggested in a causal relationship but there is no proof to warrant these opinions The disease has been observed to come on often enough after prolonged mental strain or severe psychic insult so that many believe in a neurogenic or psychogenic factor Vasomotor toxic and metabolic disturbances have also been variously blamed Familial and conjugal outbreaks of lichen planus have been encountered but are distinctly unusual

Pathology—The histopathology of lichen planus is characteristic and is of the greatest diagnostic value in doubtful cases Essentially the changes consist of a thickening of the entire epidermis especially well marked in the stratum granulosum and a dense round cell infiltration in the upper cutis The infiltrate is arranged as a narrow band paralleling the epidermis and sharply demarcated below from the mid cutis It extends over the basement membrane of the epidermis which has lost its clean line of separation from the cutis and with which it seems to blend The mucous membrane lesions have a similar pathology which allows differentiation from clinically similar eruptions of the mouth

The Eruption—The lichen planus papule is a very characteristic lesion Its diameter varies from a fraction of a millimeter to as much as a centimeter but averages 3 or 4 mm It is polygonal in outline and not rounded having a number of flattened sides It is slightly elevated usually less than 1 mm and its upper surface is flat at times with a slight central umbilication The surface is smooth and shiny and the color is dull red to purplish A violaceous hue is often present Ordinarily no perceptible scale is observed When the lesions involute a brown pigmented macule frequently remains but clears up in time

The lesions are numerous and tend to be distributed in certain areas

DIFFERENTIAL DIAGNOSIS OF

Enanthems Involving Oral and Buccal Surfaces

Eruptions involving oral and buccal mucous membranes may represent local processes or manifestations of profound systemic disturbances. Recognition of the general characteristics of the individual lesion may enable dentist or practitioner to arrive at a correct diagnosis.

DIAGNOSTIC FEATURES

Erythema

Infectious stomatitis without other manifestations. Scarlet fever with exanthem and Stoll's reaction. Charlton blanching reaction. Secondary syphilis with positive darkfield findings and serology.

Vesicles and Bullae

Infectious stomatitis. Aphthous stomatitis. Herpes simplex and herpes zoster without manifestations elsewhere. Contact dermatitis from cosmetics or dentifrices. Dermatitis medicamentosa from drugs. Erythema multiforme pemphigus and dermatitis herpetiformis with cutaneous dermatoses. Varicella and varicella with fever and exanthem. Foot and mouth disease with extremity lesions.

Macules and Papules

Fordyce's condition and leukoplakia without manifestations elsewhere. Lichen planus with exanthem. Measles with Koplik spots and exanthem.

Tumor

Epulis in pregnancy. Oropharyngeal cysts with x-ray changes and fluid by diagnostic puncture (p. 1714). Oropharyngeal neoplasms clarified by biopsy (p. 1656). Gummas with positive serology and therapeutic response to iodide.

Petechiae and Purpura

Scurvy with therapeutic response to ascorbic acid. Vitamin K deficiency with therapeutic response to menadione. Leukemias and thrombocytopenic purpura with pathognomonic hemograms (p. 3704). Subacute bacterial endocarditis with positive blood culture.

Telangiectasis

Hereditary telangiectasis with similar lesions elsewhere and normal hemogram.

Ulcers

Aphthous stomatitis without lesions elsewhere. Scurvy with therapeutic response to ascorbic acid. Vitamin B deficiencies with therapeutic response to thiamine, riboflavin or niacin. Erythema multiforme, lupus erythematosus and dermatitis herpetiformis with cutaneous exanthem. Ulceromembranous stomatitis or gingivitis with Vincent organisms demonstrable on smears (p. 50). Epithelioma or carcinoma with positive biopsy. Tuberculosis with acid fast organisms demonstrable in exudate or tissue (p. 50). Diphtheria with membranous exudate and characteristic organisms in smears and cultures (p. 302). Primary or secondary syphilis with positive darkfield and/or serology. Tertiary syphilis with therapeutic response to iodide. Agranulocytosis and leukemia with characteristic hemogram (p. 3704). Typhoid fever with positive blood culture or elevated agglutinin titer (p. 60).

| | |
|--|--|
| Papulonecrotic tuberculid | Necrotic papules with crusting and ulceration. Later scar formation. Appearing on extensor surfaces of children and young adults. A tuberculid. Get biopsy (p. 3258). |
| Pityriasis rubra pilaris | Papules surrounding hair follicles. Nutmeg-like induration. Black cored papules of dorsum of hand. Note effect of vitamins (p. 3412). |
| Prurigo milis | Allergic dermatosis with papules and wheals on extensor aspects. Look for offending allergen (p. 3343). |
| Rosacea like tuberculid of Lewandowsky | Papular eruption appearing on outer third of face in women. A tuberculid. Get biopsy (p. 3270). |
| Syphilis | Particularly copper-colored macules and papules. Often on flanks, palms and soles. Split papules at corners of mouth. Moist papules on genitals, thighs in perineal region and on mucosal surfaces. Check darkfield as well as serology (p. 45). |
| Tuberculosis verrucosa cutis | Elevated papules and pustules of hands. Seen in adults, particularly pathologists. A tuberculid. Get biopsy (p. 3258). |
| Xanthomas | Yellow papules and nodules. Often associated with diabetes mellitus or systemic reticuloendothelioses. Get blood chemistry, hemogram and bone marrow smears (p. 3244). |

of predilection. Usually they remain discrete although sometimes as they enlarge peripherally they coalesce and form large patches. Papules often spring up along the line of scratch marks (*Koebner phenomenon*) resulting in a characteristic linear arrangement of lesions. The favored sites are the *flexor* surfaces of the *wrists*, the *forearms* and the *legs* just above the ankles. The eruption may remain confined to these locales but it is not uncommonly distributed in a wider fashion. The *trunk* is often affected as well as the *arms*, *thighs* and *glans penis*. Lesions on the *palms*, *soles*, *face* and *scalp* do occur but are relatively rare (Fig. 994).

Itching is a common and often a very disturbing symptom. However it is not present in all cases.

Clinical Variants—Besides the ordinary and most usual form of lichen planus a great many variants are seen.

Lichen planus of the mucous membranes is often seen in association with the skin lesions but may exist alone. The buccal mucosa is the commonest site, especially the mucosa of the cheeks where the lesions appear as very slightly raised, smooth, whitish spots or lines. A reticulated or mosaic pattern due to criss crossing of the linear lesions is frequent. Whitish, adherent plaques may be present on the tongue, the lips and the gingivae. Lichen planus lesions may appear alone on the *glans penis* and the *labia majora* or in conjunction with skin lesions. On the *glans penis* the lesions commonly have an annular configuration while on the *labia majora* they form whitish patches which simulate leukoplakia and kraurosis vulvae.

Vesicular and bullous lichen planus is rare. These are instances in which typical lichen planus papules are present, some of which have a vesicle or bulla surmounting their surface. It is the opinion of some observers that

DIFFERENTIAL DIAGNOSIS OF

Localized Maculopapular Eruptions

See Generalized Maculopapular Eruptions p 412

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|---|---|
| Acne rosacea | Erythematous nodules with telangiectasias On middle third of face (p 3357) |
| Acne varioliformis | Papulopustules of face of adults followed by necrosis and scarring (p 3356) |
| Acne vulgaris | Common dermatosis of adolescence Comes on with papulopustules in recurrent crops (p 3358) |
| Bowen's disease | Eroded papules of trunk and extremities A malignancy of the elderly (p 3225) Get biopsy |
| Chancroid | Genital papule followed by ulceration and lymphadenopathy A venereal infection identified by demonstration of <i>H. ducreyi</i> (p 288) Exclude syphilis by darkfield microscopy |
| Dermatomycoses | Papules followed by granuloma formations and ulceration as the result of deep fungous infections Make local smears and cultures for identification of pathogen (p 3292) |
| Dermatosis papulosis nigra | Congenital dermatosis with pigmented papules appearing on middle third of face of Negro women (p 3150) |
| Erythema multiforme | Systemic dermatosis with variegated lesions usually involving dorsum of hands and feet and extensor surfaces of forearms and legs Appears in spring or autumn between the ages of 10 and 30 Note constitutional symptoms and association of nodules papules vesicles bullae and mucosal lesions Seek of feeding allergen often bacterial (p 3374) |
| Idiopathic multiple hemorrhagic sarcoma | Kaposi's disease of elderly males Purplish nodules and papules on feet and ankles A malignancy Get biopsy (p 3226) |
| Keratosis follicularis | Follicular papules covered with greasy crusts Probably a deficiency of vitamin A (p 3153) |
| Lupus miliaris disseminatus faciei | A tuberculid characterized by brownish papules appearing on the face of adults Get biopsy (p 3263) |
| Lupus vulgaris | Apple jelly nodules appearing on middle third of face A tuberculid Get biopsy (p 3263) |
| Macular atrophy | Coin sized macules of atrophic skin Often syphilitic Get Wassermann (p 3403) |
| Miliaria | The appearance of red papules and vesicles with itching and burning Due to elevation of temperature (p 3171) |
| Nevi | Pigmented and non pigmented moles (p 3204) Excise if possible |

generalized form needs differentiation from *exfoliative dermatitis* (p 3383) of other varieties and tissue study is usually necessary

Lichen planus like (lichenoid) eruptions may result from cutaneous hypersensitivity to certain drugs especially the trivalent *arsphenamines* and the *gold salts* (p 2922) It has been claimed that *bismuth subsalicylate* can also evoke a similar eruption but this is most extraordinarily rare The appearance of such a lichenoid eruption in the course of gold or arsenical therapy demands cessation of administration of the drug If doubt exists as to whether the eruption is a coincidental lichen planus or a lichenoid drug rash histopathologic study as well as careful continued observation will usually clarify the situation

Treatment—The treatment of lichen planus is nonspecific but often of considerable value It is now recognized that remissions and recrudescences are not as uncommon as previously believed The condition may persist for years with little or no change for the better or it may clear up completely and then relapse Recurrences after a period of freedom for several years are not unknown

Systemic Treatment—General hygienic measures should be advised These include the relief of nervous tension an adequate amount of rest and in some instances a vacation period Sunshine and a complete well balanced diet with added vitamins may be helpful

A variety of drugs for systemic use has been suggested The multiplicity of remedies suggests that none is of any specific or uniform value *Arsenic mercury* and *bismuth* have been used therapeutically with a variable degree of success *Arsenic* may be given in the form of the solution of potassium arsenite (Fowler's solution) in the usual ascending dosage system It may be used also by injection as 2 per cent sodium arsenate with 1 per cent phenol in sterile water (*Pollitzer's solution*) Therapy is begun with 0.3 cc subcutaneously increasing the dose by 0.1 cc at each visit until the maximum dose of 2.0 cc is being used Injections may be given every five to seven days A proprietary preparation of *mercury salicylarsenate* (*Enesol*) is often administered intramuscularly in 1 to 2 cc doses once or twice weekly

Mercury is given as the *protiodide* in pill form in doses of $\frac{1}{4}$ to $\frac{1}{2}$ grain three times daily by mouth It may be given intramuscularly as a 1 per cent solution of *bichloride of mercury* in normal saline The dose is 1 cc weekly for ten injections followed by a period of rest *Bismuth* is generally used as the *subsalicylate* as in syphilis and is given in doses of 1 cc intramuscularly every week

The administration of the *trivalent arsphenamines* as a therapeutic procedure should be discouraged It is not as beneficial as inorganic arsenic and there is always fear of serious complication One of the most intense examples of postarsphenamine jaundice ever observed appeared in a man who received three injections of neoarsphenamine for a mild lichen planus

Recently the injection of *liver extract* and *high vitamin therapy* (vitamin B complex) have been claimed to bring about satisfactory results

Local Treatment—Local treatment includes the application of topical remedies and *roentgen therapy* A great variety of local applications are used The *Lotion of Calamine and Zinc* with 1 per cent Ihenol and 5 per cent *Liquor Carbonis Detergens* is useful *Salicylic Acid* 1 to 3 per cent

these are accidental lesions possibly due to drugs (arsenic) and not inherent in the primary disease

Hypertrophic lichen planus is a fairly frequent variant usually affecting the lower extremities. It has been noted rarely on the upper extremities. The eruption is chronic and refractory and is revealed as a considerable group of isolated and coalescent papules and plaques of large size. They are roughened, horny, often verrucous, grayish to dirty brown in color and scaling. Itching is prominent and severe and it may be that the trauma of scratching and the dependent location (legs) contribute to the hypertrophic change.

Annular lichen planus is a not uncommon form in which there is a ring of typical papules, the central area being composed of normal skin. While they may be seen in any location, the glans penis is a preferred situation for these annular lesions.

Atrophic lichen planus is a rare variety in which the involution of the lesions is followed by atrophy. There result small whitish smooth atrophic macules, sometimes revealing a few dark gray or black horny plugs. These are most often seen on the trunk, especially the upper back.

Linear lichen planus refers to the form in which the papules are arranged in a linear or bandlike fashion. At times such a distribution may be unilateral on the trunk or an extremity, thus simulating the distribution of herpes zoster (zosteriform lichen planus).

Exanthematic or generalized lichen planus is fortunately an extremely rare condition in which there is generalized distribution of the eruption. The skin surface has the appearance of an exfoliative dermatitis. The eruption may appear abruptly, spread rapidly and cover the body in a few days. More often there is a history of chronic localized lichen planus which suddenly becomes converted into the generalized type.

An uncommon form is the so called *lichen planus et acuminatus atrophicans* in which more or less typical lichen planus papules may appear on the body accompanied by small patches of atrophy with alopecia of the scalp. More often the lesions of the glabrous skin have a follicular distribution and have the form of spiny follicular papules not at all characteristic of lichen planus. The histopathology is however characteristic of the disease.

Diagnosis—The diagnosis of the typical example of lichen planus is usually simple. The characteristic flat topped violaceous shiny discrete papules in their customary location, often accompanied by lesions within the mouth, present no difficulty. When the mucosal lesions exist alone differentiation from *leukoplakia* (p. 3213) may be required. The latter occurs more anteriorly as a rule, near the commissures of the mouth and on the dorsum of the tongue and is more infiltrated. Histopathologic study may be necessary.

The hypertrophic variety may resemble localized *neurodermatitis* (p. 3343) but it is uncommon for the latter to be bilaterally symmetrical or as hypertrophic as lichen planus. The presence of the typical dry papules as well as the absence of vesicular or bullous lesions serves to differentiate this variant from *eczema* or *dermatitis herpetiformis* (p. 3371). Biopsy may be needed in the atrophic form to distinguish it from *morphea* (p. 3429) but the latter consists usually of a single somewhat larger lesion. The

cases In these cases strong salicylic acid pastes and ointments from 10 to 30 per cent may also give satisfactory improvement Generalized irradiation with *ultraviolet light* may be of value

Lesions within the mouth should not be treated with silver nitrate or other caustics The metals described above or the vitamins may be employed Generally the results are poor The use of roentgen rays or radium locally is inadvisable since these may lead to serious local tissue changes In short lichen planus of the oral mucous membrane is a banal asymptomatic condition which probably never acts as a precancerosis and should therefore be treated with judicious neglect

LUPUS ERYTHEMATOSUS (CHRONIC)

Lupus erythematosus is a *systemic disease* which may pursue an acute subacute or chronic course It is characterized by cutaneous lesions which may be localized or disseminated

Clinical Manifestations—Chronic lupus erythematosus occurs as a localized chronic discoid variety and in a disseminated form The relationships of the two types is not clear Some authorities consider them variants of the same disease while others regard them as distinct and unrelated entities Since acute dissemination may occur in patients with the chronic discoid variety the weight of opinion favors an identical origin

Chronic Discoid Lupus Erythematosus—The localized form is the commonest variety of lupus erythematosus It affects white persons far more often than Negroes or other dark skinned individuals Women are more susceptible although the disease is not at all uncommon in men It is most frequent between the ages of twenty and fifty years

Disseminated Chronic Discoid Lupus Erythematosus—When the lesions of the chronic discoid variety appear on other parts of the body as well as the head the condition is spoken of as disseminated chronic discoid lupus erythematosus The eruption may occur on the chest arms or hands The patient remains in excellent health and has no complaints other than the slight itching and burning of the rash

The *progress* of the affliction is extremely slow with a tendency to *remissions* and *exacerbations* especially after exposure to sun In the very superficial forms little or no atrophy may result but in most cases an atrophic white spot appears and remains permanently The prognosis is generally excellent except for the occasional instance in which the lesion becomes acutely disseminated

Etiology—The cause of the condition is unknown although it is usually accompanied by *photosensitivity* It may follow exposure to the *sun* or *ultraviolet light* and is as a rule aggravated by these factors It was formerly thought to be closely related to *pulmonary tuberculosis* but this concept has been generally discarded Many believe that it is a syndrome of multiple etiology in which various bacterial agents (notably streptococci) act as a source of *toxins* to the skin Perhaps the toxic agents sensitize the tissues to light and the latter then acts as the precipitating agent Chronic mild *metallic poisoning* especially with lead has been suggested as a possible cause

Clinical Appearance—The *exposed surfaces* are usually affected especially the malar eminences the nasal bridge the ears the scalp and the

in ointment or paste may cause a reduction in size of the lesions The various tar preparations are helpful in the chronic cases as 2 per cent

DIFFERENTIAL DIAGNOSIS OF

Telangiectases

See also Petechiae p 3398 Purpura p 3422

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|--|---|
| Acne rosacea | Chronic dermatosis of face with redness and telangiectases (p 3357) |
| Adenoma sebaceum | Hereditary dermatosis of childhood Note yellow and red papules of middle third of face with telangiectases Associated with nervous and mental disturbances due to tuberous sclerosis (p 3148) |
| Epithelioma | Of basal or squamous cell varieties Ulcerated nodules with rolled border of face hands or genitals Get biopsy (p 3220) |
| Lupus erythematosus | Systemic disturbance of females with widespread involvement of vessels A systemic angitis Note photosensitivity Facial or general dermatosis often with butterfly distribution over bridge of nose (p 3395) |
| Multiple benign cystic epitheliomas | Rare familial disease of females Lentil sized cysts over face Get biopsy (p 3208) |
| Multiple hemorrhagic familial telangiectases | Hereditary and familial disease Multiple lesions of skin and internal organs Note telangiectases of cutaneous or mucosal surfaces. Associated with frequent hemorrhages (p 1119) |
| Nevus | Of spider variety with multiple dilated capillaries radiating from the central elevated vessel Destroy by electrocoagulation, if possible |
| Poikiloderma atrophicum vasculare | A rare dermatosis involving flexural regions Characterized by pigmentation papule formation telangiectases and atrophy (p 3413) |
| Radiodermatitis | Atrophy or hyperkeratosis of the skin with telangiectases A precancerous lesion Get biopsy (p 3177) |
| Sarcoidosis | Systemic disorder probably associated with tuberculosis Note brownish nodules or plaques especially on face and extremities Often associated with parotid and uveal lesions. Get biopsy (p 3271) |
| Xeroderma pigmentosum | Congenital dermatosis with abnormal photosensitivity of exposed parts Lesion characterized by pigmented macules atrophic spots telangiectases and keratoses A precancerous Get biopsy (p 3158) |

Crude Coal Tar in equal parts of Lanolin and Petroleum or in Ointment of Zinc Oxide

Röntgen therapy may give prompt results or may be completely without benefit It is often of particular value in the refractory hypertrophic

cases In these cases strong salicylic acid pastes and ointments from 10 to 30 per cent may also give satisfactory improvement Generalized irradiation with *ultraviolet light* may be of value

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Clinical Appearance—The *exposed surfaces* are usually affected especially the malar eminences the nasal bridge the ears the scalp and the

lips particularly the lower. The lesions consist of sharply defined rounded or irregular patches which develop slowly and progress with subtle chronicity. A solitary lesion may vary in size from 1.0 to 4.0 cm and consist at its inception of a slightly raised well demarcated area of redness firmer than the normal tissues and partly covered by a dry adherent scale. When the scale is forcibly removed its undersurface shows keratotic prolongations which fit into visibly dilated orifices of hair follicles or sweat ducts (Fig. 995).

The lesion enlarges peripherally at an extremely slow rate and as it does so the central portion tends to become *whitened* and *atrophic*. Telangiectatic vessels may be present over and above the plaque of eruption. When the disease has lost all its activity the area becomes converted into a white atrophic slightly depressed *macule* which is irrevocable. On the scalp the atrophic process destroys the hair follicles and results in a circumscribed blanched area of permanent *alopecia*.

The chronic discoid form may exist as an isolated *solitary plaque* or there may be a number of coalescent lesions present. On the face there is a tendency for simultaneous involvement of the malar eminences and the nasal bridge producing the so called *butterfly pattern*. On the lip or buccal mucosa the lesion is a sharply defined slightly elevated reddish area with whitish atrophic points or lines. The border is dusky red and is traversed by telangiectatic vessels. At times there may be some erosion or even superficial ulceration within the area.

Diagnosis—The chronic discoid form of lupus erythematosus is unmistakable in its typical distribution on the face and its morphologic constituents of erythema, adherent scale, keratotic plugs, dilated follicular orifices, telangiectases and atrophy. The disseminated variety may be confused with *acute disseminated lupus erythematosus* (p. 3399). However the latter is a systemic disease with widespread visceral manifestations.

Treatment—Treatment is often but not always highly successful. The patient must be warned against exposure to sunlight, ultraviolet light and roentgen treatment which may exacerbate the condition and has even been known to initiate acute dissemination. The use of *light protective creams* and *lotions* (p. 3140) is advisable. The removal of foci of infection particularly in the teeth and tonsils has been known to produce considerable benefit. Improvement of the general health is always in order including the administration of anti-anemic drugs and a well balanced diet with good vitamin content and additional multivitamin preparations.

As in other obscure dermatoses many systemic remedies are reported as efficacious casting doubt on the specificity of each and all. The most dangerous and seemingly the most successful drug is *gold* injected as *gold sodium thiosulfate*. Treatment is initiated with an intravenous dose of 5 mg. This amount is increased 5 mg. weekly until a maximum amount of 25 mg. is injected. Treatments are given weekly for 20 injections provided that toxic symptoms (p. 2922) do not develop. In refractory cases and with absence of toxicology the dose may be increased by 5 mg. increments to 50 mg. *Sulfanilamide* and *sulfapyridine* also have been used with passing benefit in the chronic discoid form. The danger of photosensitization from sulfanilamide and general toxic effects would seem to make other procedures more desirable.

Quinine may be tried by mouth over extended periods and in the *Holander* treatment the lesions also are painted once daily with *tincture of iodine*. Improvement is very slow and in fact often incomplete or absent.



Fig 994—Lichen planus of forearm and of penis



Fig 995—Lupus erythematosus



Fig 996—Lupus erythematosus Chronic fixed discoid variety

Bismuth is used most often as the subsalicylate as in syphilis and given in doses of 1 cc (0.13 gm of the salt) weekly by intramuscular injection. This procedure is ordinarily innocuous but improvement occurs very

DIFFERENTIAL DIAGNOSIS OF

Petechial Dermatoses

See also Telangiectases p 3394 Purpuras p 3422

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

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| Acute disseminated lupus erythematosus | Fatal systemic vascular disorder of the female Lesions of cheek and nose Associated with dusky red edema and ulceration Get biopsy (p 3399) Note hypersensitivity to sunlight |
| Anaphylactoid purpura | Systemic allergy often associated with abdominal and joint symptoms Seek offending allergen often bacterial (p 3424) |
| Drug eruptions | Dermatitis medicamentosa Particularly due to sulfonamides gold, arsphenamine and silver Check history |
| Gonococcemia | Bacteremia following venereal infection (p 219) Get blood culture Start intensive penicillin therapy |
| Haverhill fever | A variety of rat bite fever with generalized maculopapular eruption (p 363) Start intensive penicillin therapy |
| Leukemia | Fatal disease of blood forming organs Get hemogram and marrow smear (p 1100) |
| Meningococcemia | Bacteremia with generalized purpuric eruption and later evidences of meningeal irritation Get blood culture and examine spinal fluid (p 211) Start intensive parenteral and intrathecal therapy with penicillin |
| Plague | Epidemic infection with <i>P. pestis</i> May be bubonic or pneumonic (p 321) |
| Idiopathic thrombocytopenic purpura | Bleeding tendency with diminution of blood platelets Consider splenectomy (p 1116) |
| Rheumatic fever | Rarely associated with petechiae unless there is a superimposed subacute bacterial endocarditis (p 186) Get blood culture Start intensive combined therapy with penicillin and heparin (p 106) |
| Rocky Mountain spotted fever | Rickettsial infection following tick bite Note centripetally advancing macular rash which later becomes hemorrhagic Check Weil-Felix reaction (p 376) |
| Scurvy | Hemorrhagic disorder associated with deficiency of vitamin C Note therapeutic response to ascorbic acid (p 2857) |
| Smallpox | Monomorphic papular vesicular and pustular eruption first involving face and extensor surfaces May be hemorrhagic with petechiae in malignant instances (p 424) |
| Streptococcemia | Bacteremia with variegated eruptions Get blood culture (p 163) Start intensive penicillin therapy |
| Subacute bacterial endocarditis | Most frequent of petechial disorders Lesions engrafted on congenital valve defect or rheumatic endocarditis Get blood culture (p 286) Start intensive combined treatment with penicillin and heparin (p 1) |

Typhus fever

Rickettsial infection with centrifugally advancing macular eruption that later becomes hemorrhagic. Check Weil-Felix reaction (p. 369).

Vitamin K deficiency

Hemorrhagic disorder due to hypoprothrombinemia in newborn and in the jaundiced. Note therapeutic response to menadiol (p. 1111).

slowly and it yields a distinctly lower percentage of cures than the gold salts. Favorable results have recently been reported from the administration of oral bismuth (Sobisminol) 3 to 6 capsules daily over long periods of time.

Bismarsen (bismuth arspenamine sulfonate) has been recommended for stubborn examples. The usual dose is 0.1 to 0.2 gm in 1 to 2 cc of sterile water given intramuscularly at weekly intervals. This drug may cause any of the complications seen with the arspenamines (p. 116).

In some cases where the discoid lesion is distinctly thickened, hypertrophic, almost verrucous, the direct use of *carbon dioxide snow* on the lesion or light *electrodesiccation* may be of considerable value.

ACUTE DISSEMINATED LUPUS ERYTHEMATOSUS

Acute disseminated lupus erythematosus is seen in females between puberty and the menopause. The highest incidence is in the age period from 12 to 30 years, instances becoming less and less frequent as patients advance into the thirties and forties. The disease is infrequent in Negroes. The cause is unknown and while the clinical impression is that of an acute overwhelming sepsis, blood cultures are sterile.

Etiology and Pathogenesis.—The belief that *tuberculosis* is a factor of importance has become less prevalent with increased study and investigation. Various bacterial agents have been blamed, especially streptococci, either as the direct cause or through toxic or allergic mechanisms. Sensitization to normal or abnormal metabolic products has also been put forward as a possible factor. *Endocrine dysfunction* may be involved, especially in view of the great prevalence of the disease in females. The importance of *photosensitivity* and the *gold salts* as precipitants is doubtful. *Exposure to cold* may also act as a precipitant since the syndrome may arise during the winter when light sensitivity does not apparently play a significant role. Onset during *pregnancy* is not an uncommon occurrence.

The prevailing opinion is that there exists in these individuals an abnormal sensitivity of the small peripheral blood vessels or collagenous tissues to a variety of stimuli which act upon them to bring on the characteristic syndrome. The pathologic process, according to one theory, apparently begins in the collagenous tissues and intercellular cement substances, the vascular endothelium being secondarily affected. This process is not unique since a similar state exists in rheumatic fever, scleroderma and periarthritis nodosa.

Pathology.—Examination of the diseased tissues reveals a variety of alterations which include *capillary dilatation*, *extravasation of blood and serum*, *proliferation of the capillary endothelium*, *thrombi*, and *degenerative and necrotic lesions of the capillaries*. A characteristic finding is widespread *fibrinoid degeneration of the collagen*. In the affected tissues the glomerular vessels of the kidneys show hyaline thickening and the characteristic wire-loop change.

Clinical Manifestations.—The disease is a systemic entity in which the cutaneous manifestations constitute a minor facet of the entire syndrome. In fact the entire syndrome may be present without the characteristic skin lesions.

Skin Lesions—The eruption consists of a dusky red or livid edematous lesion distributed in 'butterfly pattern' over the *cheeks* and *nose*. There is a close resemblance to *erysipelas* (p 167). The lesion is very superficial and consequently lacks the features of the chronic discoid variety unless they were previously present.

The facial lesion may be localized or it may spread to involve the entire face, the ears and the neck. Similar edematous erythematous patches may be present on the chest, the upper extremities and the fingers. *Telangiectases* may appear on the face and the hands especially over the terminal phalanges and under the nails. *Purpuric* and *petechial* lesions are common. *Mouth eruptions* are often present and include bullous, erosive and ulcerative lesions. Patchy or diffuse toxic alopecia of the scalp is frequent.



Fig 997—Acute disseminated lupus erythematosus with rapidly fatal termination. (Courtesy of Dr H P Jacobson.)

Systemic Symptoms—Fever is mild at the outset but becomes higher and more septic in type as the toxicity increases and the terminal stage is approached. *Arthralgias* and actual *arthritis* are commonly present, often preceding the actual onset of the disease. *Polyserositis pericarditis* and *pleuritis*, sometimes with effusion, are frequent. *Fundus hemorrhages* and *exudates* are found in about one third of the patients. *Renal involvement* is present in every sufferer although of variable severity. There may be enlargement of the *superficial lymph nodes* and moderate *splenomegaly*. *Cardiac murmurs* may be heard.

Libman Sacks Syndrome—The Libman Sacks syndrome resembles closely acute disseminated lupus erythematosus. In the latter disease *endocarditis* is not regularly present while it is a constant feature of the Libman Sacks syndrome. Mural and valvular endocardial vegetations, bacteria free, are found at autopsy. In both the blood cultures are sterile.

* Andrews Diseases of the Skin

Considerable confusion still exists as to the relationship of Libman Sacks syndrome acute disseminated lupus erythematosus and similar conditions described under other titles. One thing is certain that the entire clinical picture of acute disseminated lupus erythematosus may occur without the characteristic cutaneous lesions. More and more students of the subject are leaning to the belief that Libman Sacks syndrome is not a precise entity but that it includes examples of acute disseminated lupus erythematosus and related conditions.

Laboratory Findings—The laboratory findings include secondary anemia leukopenia and thrombocytopenia and evidences of bone marrow depression. Patients have been observed in whom the syndrome of *thrombocytopenic purpura* (p 1116) preceded the appearance of the characteristic picture of acute disseminated lupus erythematosus by several months. When the thrombocytopenia is marked during the course of the illness the purpuric manifestations may dominate the picture. They are not constantly present nor need all appear simultaneously in the individual patient. Renal damage may be indicated by *albuminuria* microscopic *hematuria* the presence of *casts* and moderate to marked degrees of *azotemia*. The blood culture is sterile although a variety of organisms may be found in the premortal phase. These are most likely secondary invaders and not etiologically important.

Course—The disease may begin abruptly with severe constitutional symptoms and run its course in four to eight weeks. It may be subacute with milder symptoms and periods of remission going on this way for several years before death ensues. Authentic cases of complete recovery are rare.

Diagnosis—Acute disseminated lupus erythematosus must be differentiated from acute *sepsis* (p 19) *exudative erythema multiforme bullosum* (p 3376) and *dermatomyositis* (p 3373). The age and sex of the patient the morphology and distribution of the eruption and the concomitant visceral and laboratory findings usually serve to distinguish the conditions but the opinion of a specialist dermatologist is advised.

Prognosis—The prognosis is extremely poor complete recovery being extremely rare. The actual cause of death may be *pneumonia toxemia septicemia uremia* or *pericarditis with effusion*.

Treatment—There is obviously no satisfactory therapy. The patient should be hospitalized in a *darkened room* to avoid the aggravating effects of light. Since the condition is often activated by injections of tuberculin serums vaccines and removal of foci of infection none of these procedures should be performed. Repeated small *blood transfusions* (200 to 300 cc) are valuable in improving the resistance of the patient.

A variety of remedies has been recommended including *autohemotherapy* *quinine* *liver extract* *testosterone propionate* *nicotinamide* in large doses *bismuth* and *gold*. The last is contraindicated according to most observers although some claim that it is beneficial when used in doses of 1 or 2 mg.

Recently successful results have been reported from the administration of *sulfanilamide* although the photosensitizing action of this drug cannot be disregarded. The use of other members of the sulfonamide group is under study.

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|-----------------------|---|
| Thiamine deficiency | Peripheral neuritis and postneuritic atrophy or edema (wet beriberi). Note response to vitamin B extract (p. 618) |
| Xeroderma pigmentosum | Pigmented macules telangiectases keratoses atrophy and precancerous changes of exposed part, with abnormal photosensitivity (p. 3158) |

MACULAR ATROPHY

Macular atrophy is a descriptive term referring to patients who exhibit circumscribed coin sized areas of atrophy. The primary type (*anetoderma erythematodes*) is of unknown etiology and the atrophic phase is preceded by an erythematous macule. White atrophic spots may be present concurrently with reddened macules which are in the process of involution into atrophic areas.

Secondary macular atrophy is most frequently the result of *sypilis*. Apparently in a small number of patients who develop a secondary syphilitic dermatitis there is destruction of the elastic and collagenous tissue. Months



Fig. 938.—Macular atrophy due to syphilis

or years later there are found white atrophic macules which may bulge slightly but can be depressed into the skin with the finger. Palpation suggests the presence of a hernial ring. The lesions are most common upon the trunk in both primary and secondary macular atrophy. However a widespread distribution including the extremities may occur.

There is no effectual therapy.

MILIUM

Milia are common lesions about the *eyelids*, *forehead*, *cheeks*, *temples* and *genitals*. They are most frequently observed in *young persons* as an idiopathic condition. They are sometimes associated with *acne*, *pemphigus* and *epidermolysis bullosa*.

The Lesion.—Milia are small spherical masses of *sebum* and *epithelial detritus* retained in the skin and covered by intact epithelium. They appear as rounded slightly raised white masses somewhat smaller or larger than a pinhead. In rare instances they have been known to undergo calcific changes (cutaneous calculi). They are distinguished from comedones from which a sebaceous plug can be expressed.

DIFFERENTIAL DIAGNOSIS OF

Atrophy of the Skin

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

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|------------------------------------|--|
| Acrosclerosis | Atrophy of fingers and toes especially in Raynaud's disease (p 3367) |
| Actinic dermatitis | Atrophy thickening and pigmentation of skin after prolonged exposure to ultraviolet |
| Arteriosclerosis | Atrophy thinning and ulceration especially of skin of feet in peripheral vascular disease |
| Causalgia | Pain and atrophy of skin after destruction of peripheral nerve (p 3229) |
| Dermatomyositis | Edema tenderness and atrophy of skin during course of systemic disorder Get biopsy (p 3373) |
| Folliculitis decalvans | Circumscribed pustules of scalp with scarring and permanent alopecia (p 3442) |
| Leprosy | Nodular and ulcerative lesions with thickening of nerve trunks anesthesia and post-neuritic atrophy Get biopsy (p 273) |
| Lupus erythematosus | Patches of redness and scaling Often of middle third of face (butterfly) Projecting keratotic plugs Photosensitivity Get biopsy (p 3395) |
| Lupus vulgaris | Apple jelly nodules of middle third of face A tubercloid Get biopsy (p 3263) |
| Macular atrophy | Coin sized areas of atrophy Often syphilitic Get Wassermann (p 3403) |
| Necrobiosis lipoidica diabeticorum | Of anterior aspect of thigh Hypersensitivity to insulin injections (p 3240) |
| Neuritic atrophy | Glossy whitened area along distribution of involved sensory nerve (p 3228) |
| Pituitary basophilism | Purplish abdominal striae With endocrine disorder associated with hirsutism hypertension and buffalo type of obesity (p 1162) |
| Poikiloderma atrophicum vasculare | Pigmented, lichenoid papules and telangiectases of flexural regions (p 3413) |
| Pregnancy | Striae of abdominal wall |
| Progressive facial hemiatrophy | Asymmetry due to atrophy of bones cartilages and underlying soft parts (p 2834) |
| Radiodermatitis | History of x ray or radium treatment followed by atrophy or hyperkeratosis with telangiectases (p 3177) |
| Sarcoidosis | Brownish nodules or plaque often on face Probably a tubercloid Often associated with ocular lesions Get biopsy (p 3271) |
| Sclerema | Of newborn Generalized whiteness and shrinking of skin May be fatal (p 3157) |
| Scleroderma | Localized or diffuse atrophy with production of hide bound skin (p 3427) |
| Senile atrophy | Thinning wrinkling and dryness of skin. |
| Senile vulvovaginitis | Fissures leukoplakia pruritus dyspareunia atrophy and precancerous changes Try effects of estrogen (p 2515) |

This is probably the result of washing with soap and water and consequent desquamation and expulsion of the milium. If many lesions are present desquamation by *cold quartz light* or 5 per cent *salicylic acid* in 70 per cent alcohol may be effective. Undoubtedly the simplest procedure is to nick the skin over the milium with a sharp needle or a very fine cataract knife and expel the mass with a comedone extractor. Healing occurs without the production of a scar.

PANNICULITIS (RELAPSING FEBRILE NONSUPPURATIVE)

Relapsing febrile nonsuppurative panniculitis is a very rare disorder characterized by the appearance of recurrent crops of tender painful *subcutaneous nodules* in association with mild systemic symptoms. The cause is unknown but most observers believe the syndrome is a *subcutaneous fat necrosis* with foreign body inflammatory reaction evoked by different noxious substances. *Infectious agents* and *drugs* have been held responsible. There is no disturbance of the fat metabolism. The condition has been seen from youth to adult life.

The *characteristic nodules* are generally multiple of variable size and are raised. The overlying skin is bluish red in color. There is gradual involution of the nodules, the very small ones leaving no sequelae, the large ones being followed by a permanent saucerlike depression. The skin over the depression is entirely normal in appearance.

Systemic symptoms usually consist of moderate fever and malaise but leukopenia and splenomegaly have been reported. Recovery is apparently the rule but *recurrences* may occur at intervals of a few months or several years.

There is no satisfactory *treatment*.

PEMPHIGUS

Pemphigus is a rare *bullous* disease of the skin and mucous membranes. It is of unknown etiology and probably always terminates *fatally*. It may pursue an *acute* or *chronic* course and its essential bullous nature may be masked by secondary changes in the skin giving it an exfoliative (pemphigus foliaceus) or a vegetating (pemphigus vegetans) character.

The disease is most frequent in adults between the ages of forty and sixty years but has been seen in much younger and older persons. Some statistical surveys show a higher incidence in males, others in females. Jews seem to be especially prone to the condition, especially those coming from Russia. Italians are next in order of susceptibility. However the disease has been observed in all races and sects and in every part of the world. In rare instances the occurrence of more than a single case in the same family has been recorded.

Etiology—The cause of pemphigus is unknown. The concept that it is due to some disturbance of the *nervous system* has not been borne out by investigation and postmortem examination does not reveal any consistent pathology of the nerve tissues. It has been suggested that the condition is a *profound toxemia* caused by some unknown noxious agent. A great variety of infectious organisms have been isolated but they must be looked upon in the present state of our knowledge as secondary invaders. In recent years efforts to impute the disease to a *filtrable virus*

DIFFERENTIAL DIAGNOSIS OF

Depigmentation of the Skin

See also Generalized Pigmentation p 3242 Localized Pigmentation p 3154

Absence of pigment from the skin may be of congenital or acquired etiology. The congenital variety is albinism whilst the acquired types are called vitiligo or leukoderma. Vitiligo refers to primary loss of pigment whereas leukoderma describes instances in which the loss of pigment is the sequel of some precedent cutaneous disturbance.

CLINICAL MANIFESTATIONS

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|---------------------------|---|
| Albinism | Congenital absence of pigment in skin hair and eyes. May be partial or complete. Iris is pink and pupil appears red due to absence of pigment from the choroid. Skin extremely vulnerable to sunlight which also produces photophobia and nystagmus. |
| Endocrinologic leukoderma | Occasionally observed in women following menopause. Often in association with small signs of anterior pituitary deficiency (p 1166). |
| Leprosy | White anesthetic macules. Get biopsy (p 273). |
| Leukoderma | Depigmentation after involvement of a dermatosis. May follow lichen planus psoriasis pityriasis rosea parapsoriasis tinea versicolor pinta and syphilis. Depigmentation after a tinea is termed achromia post parasitaria. Pinta produces permanent depigmentation (p 353). |
| Leukoplakia | White plaques with fissures. Often seen on buccal mucosa lips tongue penis or vulva. A precancerous. Get biopsy (p 3213). |
| Morphea | White patches with violaceous halo due to circumscribed scleroderma (p 3429). |
| Occupational leukoderma | As a result of exposure to hydroquinone derivatives used in rubber gloves. |
| Post neuritic | Following peripheral nerve injury. Note neurologic signs (p 3228). |
| Scleroderma | Generalized hidebound condition of skin (p 3428). |
| Striae | After pregnancy. With basophilic adenomas of pituitary gland (p 1166). |
| Tophi | White deposits of sodium urate crystals in gout. Get blood uric acid (p 2867). |
| Vitiligo | Primary acquired leukoderma. Most common in Negroes who reveal larger and smaller patches of depigmentation. Achromic areas enlarge peripherally and may coalesce. Face hands and anogenital regions most frequently involved. Exposure to sunlight intensifies achromia by darkening adjacent normal skin. |

Treatment—Women chiefly apply for treatment and many confirm the observation that some of the lesions have disappeared spontaneously.

This is probably the result of washing with soap and water and consequent desquamation and expulsion of the milium. If many lesions are present desquamation by *cold quartz light* or 5 per cent *salicylic acid* in 70 per cent alcohol may be effective. Undoubtedly the simplest procedure is to nick the skin over the milium with a sharp needle or a very fine cataract knife and expel the mass with a comedone extractor. Healing occurs without the production of a scar.

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have met with contradictory results. Some have been able to show the presence of a virus while others equally able and painstaking have found no such organism present. The evidence now at hand is insufficient to warrant the belief that a filtrable virus is the causative agent. Recent postmortem investigations of body chemistry and tissues have given rise to the theory of *adrenal damage* with insufficiency as a factor.

Clinical Manifestations—Pemphigus may appear in several clinical forms besides the common vulgaris variety.

Pemphigus Vulgaris—Pemphigus vulgaris the commonest form encountered usually commences insidiously. Constitutional symptoms are lacking at the outset and do not appear until the disease is well advanced. The first lesions most often are noted upon the trunk or in the mouth but they may appear in both sites simultaneously. There may be a solitary lesion but as a rule multiple *bullae* are present and as they dry up new ones continually appear. This process may continue without remission until the general health gradually becomes impaired and *death* follows in two to six months. On the other hand there may be complete or incomplete *remissions* lasting many months or even years followed by recrudescence. In this manner patients may survive for several years.

The characteristic lesion is the *bulla* which is rounded or oval tense and filled with clear serum. It may vary from a few millimeters to 10 cm or more in diameter. At times the clear yellowish serous content becomes cloudy or hemorrhagic. The bulla springs from healthy skin and commonly has no surrounding red halo although this may appear subsequently. The fluid may be absorbed and the bulla dry up and disappear or the bulla may rupture and become a crusted lesion followed by brown pigmentation. In some instances there is denudation of large areas of skin the appearance resembling that of an extensive second degree burn.

In the mouth the bullae rupture promptly and produce raw eroded surfaces covered with a whitish film. Similar eroded areas due to ruptured bullae may appear on the vulva and in the vagina. *Itching* of the skin is not a prominent symptom but is not infrequently present. More often the patient complains of burning of the skin, soreness and bleeding in the mouth and as the disease progresses loss of weight and weakness (Fig 999).

Pemphigus Foliaceus—Pemphigus foliaceus is a rare variant in which the bullae are small and indefinite. The entire skin surface becomes converted into an *inflammatory scaling and crusted mass*. Even when the scaling inflamed state is chiefly in evidence close inspection may reveal flaccid bullae. The retention of moisture and decomposition by saprophytes often lead to a foul odor. Denuded painful areas in the mouth and throat occur as in the common variety. The condition of the skin closely resembles that seen in *exfoliative dermatitis* (p 3383). In other respects the course and outcome are similar to those of pemphigus vulgaris.

Pemphigus Vegetans—Pemphigus vegetans is another rare variant which starts ordinarily as pemphigus vulgaris but in which the ruptured bullae are soon replaced by *exuberant warty masses*. These are raised irregular cauliflower like excrescences crusted and exuding a thin brownish malodorous discharge. While vegetations may appear in any area

they are more common in the folds of the skin the axillae neck groins perineum elbow and knee bends Lesions of the *mouth* and of the mucosa of the *vagina* also occur The course and outcome are in general the same as those of pemphigus vulgaris

Pemphigus of the Conjunctiva—Pemphigus of the conjunctiva (essential shrinking of the eyeball) is an unusual variation which may occur as an isolated solitary phenomenon but most often is associated with cutaneous and mucosal lesions The involvement is almost always *bilateral* and frequently extends to the cornea where the consequent scarring causes *blindness* Aside from this serious sequel of blindness the disease is usually benign the cutaneous and mucosal eruption rarely assuming significant proportions Apparently life is not shortened by the disease

Butcher's Pemphigus—Butcher's pemphigus although it is usually discussed with pemphigus is entirely unrelated to it It is rather an *acute infectious bullous dermatitis* It is an extremely serious often rapidly *fatal condition* which has been most often observed in butchers and others who handle animal tissues It has also been reported following vaccination and trauma to the skin especially when infection supervenes

There is a generalized eruption of *bullae* which become *purulent* and *hemorrhagic* and attack the mucosal surfaces as well as the skin The patient is seriously ill with *chills high fever* and evidences of *prostration* The general impression is that these are instances of *generalized sepsis* even though the blood culture is negative It would seem that in addition to the usual supportive measures the most helpful therapy would be one or the other member of the *sulfonamide group*

Special Tests—The *Nikolsky sign* is usually positive in pemphigus but may also be present in other diseases especially *epidermolysis bullosa* (p 3151) When the skin is pinched lightly it separates with facility or raises up in the form of a bulla

The *phytopharmacologic test* (*Pels Macht*) is based upon the belief that there is a toxic substance in the blood serum which interferes with the growth of certain seedlings (*lupinus albus*) A similar toxic substance is said to be present in *leprosy* and *pernicious anemia* but not in *dermatitis herpetiformis* or *bullous erythema multiforme* The last two conditions particularly may present a confusing diagnostic problem in which the test is useful According to Pels and Macht when the index of toxicity is below 60 per cent a presumptive diagnosis of pemphigus can be made

Diagnosis—Pemphigus vulgaris must be differentiated from the bullous form of *erythema multiforme* (p 3374) *dermatitis herpetiformis* (p 3371) and *drug eruptions* (p 3334) In infants the disease is very rare but might be confused with the *bullous syphiloderm* (p 3281) and *impetigo contagiosa neonatorum* (p 3251) These can usually be readily ruled out by the presence of collateral lesions serologic reactions of the blood and response to therapy

The most difficult problem however may arise in deciding between *dermatitis herpetiformis* *bullous erythema multiforme* and *pemphigus vulgaris* The grouping of polymorphous lesions the rarity of mouth lesions the intense itching and the maintenance of good health favor *dermatitis herpetiformis* (p 3371) In *erythema multiforme* (p 3374) the

characteristic distribution of the eruption the presence of iris lesions and the acute course with recovery are important differential points The history of drug ingestion should always suggest the possibility of a drug (especially iodides) eruption and further study usually allows of a final decision

Pemphigus foliaceus must be differentiated from *exfoliative dermatitis* (p 3383) The latter may be secondary to a known skin disease it may be of so called primary origin or it may be brought on by drug sensitization The presence of bullae a positive Nikolsky sign the absence of history of a precedent skin disease or drug administration the course of the condition and the histopathology assist in the differentiation

Pemphigus vegetans may simulate masses of *acuminate uarts* the *condylomata lata* of *syphilis* and *fungating bromodermas* or *iododermas* History darkfield examination serologic tests of the blood and response to therapy serve in reaching a correct diagnosis

Treatment—There is no single satisfactory treatment for pemphigus All therapeutic procedures must be judged in the light of the known tendency of the disease to follow at times a protracted course with periods of remission and exacerbation It is generally believed that therapy of certain kinds may bring on remission and prolong life

Preservation of the general health is of vital importance The administration of a high calory high vitamin diet is recommended When the patient is unable to take fluids because of the soreness of the mouth intravenous administration of dextrose solution is advisable Repeated small blood transfusions are a valuable supportive measure

Germanin and Moranyl—Most reports agree that the most beneficial drug is *Germanin* (Bayer 205) first used in the treatment of trypanosomiasis This is a highly toxic drug especially for the kidneys and the urine must be examined frequently especially before each injection for evidences of nephritis (albumin casts and red blood cells) If these are present the drug is discontinued The solution must be freshly prepared before each injection boiled and then cooled to room temperature before administration The usual initial dose is 0.25 gm the second dose 0.5 gm the third 0.75 gm and the succeeding doses 1.0 gm Injections are given at intervals of five days and a total dosage of 5.0 gm is not as a rule exceeded A similar preparation prepared in France (*Moranyl*) is used in the same manner

Vitamin D and Dihydratachysterol—Remissions have been obtained in some instances after massive doses of *vitamin D* (p 620) in the form of *viosterol* The recommended dose varies from 100,000 to 500,000 units daily A subproduct of ergosterol *dihydratachysterol* (p 621) may also be used This is recommended in daily doses of 10 cc orally for seven to ten days followed by lesser amounts and finally a maintenance dose of 5 to 15 cc weekly With both these drugs there is danger of hypercalcemia and renal injury The level of the serum calcium must be determined at intervals to forestall any dangerous elevations The improvement obtained is usually temporary

Sulfonamide—Sulfanilamide has been given with temporary benefit and it seems to be more effective than the other sulfonamides Amelioration does not always occur and is generally only of short duration

Arsenic—Arsenic in various forms has long been used and is a beneficial remedy at times. It may be given orally as *Fowler's solution* by injection as *sodium arsenate* or as *iron cacodylate* (p 116). *Acetarsone* orally is given in 0.25 gm tablets four times daily for four days. After a

DIFFERENTIAL DIAGNOSIS OF

The Commoner Generalized Afebrile Eruptions

Generalized eruptions associated with fever have previously been discussed. The present concern is consideration of extensive rashes in the afebrile individual. Some of these rashes as in syphilis represent a systemic infection despite the absence of disturbance of the temperature regulating mechanism; other reactions are allergic (atopic dermatitis and dermatitis medicamentosa). The most frequently encountered rashes are the descriptive dermatoses (p 3355) of pityriasis rosea, psoriasis and pemphigus.

CAUSE

DIAGNOSTIC FEATURES

Atopic Dermatitis (Eczema)

An allergic reaction, with itching erythema and vesiculation, and scaling. Frequent in infancy and childhood (p 3342).

Dermatitis Medicamentosa

Macular papular erythematous, urticarial and pustular eruptions following analgesics, sulfonamides, quinine, arsenicals, iodides, phenolphthalein, bromides, gold, salicylates, etc. (p 3335).

Exfoliative Dermatitis

Serious and often fatal peeling, often following drugs, particularly arsenicals (p 3383).

Framboesia Tropica

Extragenital crusted ulcer of primary lesion and generalized papular eruption. Wassermann and darkfield positive (p 351). Favorable response to arsenicals.

Generalized Vaccinia

Varioliform eruption following vaccination (p 433).

Molluscum Contagiosum

Virus infection causing umbilicated vesicles especially of face, trunk, and genitals.

Pemphigus

Fatal bullous eruption of skin and mucous membranes (p 3405).

Pityriasis Rosea

Herald scaling patch followed by generalized itching macular eruption of trunk, thighs and shoulders. Self limited (p 3410).

Psoriasis

Chronic recurrent scaling and itching papules with predilection for elbows, knees and extensor surfaces (p 3414).

Syphilis

Maculopapular, vesicular or papular non-pruritic lesions. Darkfield examination and serology positive (p 337). Specific responses to penicillin and arsenic.

Xerosis

Dryness of skin and papules at mouths of hair follicles (keratosis pilaris) in vitamin A deficiency (p 3234). Specific response to therapy.

period of rest for four days; this dosage is repeated. *Neocarsphenamine* and *mapharsen* have also been used.

Adrenal Cortical Extract—Because of the depression of chlorides and the possibility of adrenal insufficiency, the use of *physiologic saline solution* intravenously with *adrenal cortex extract* intramuscularly has been

suggested Where these have been employed there has been observed improvement and remission but these are only transient

Mercury—Recently there has been recommended the daily intramuscular injection of 1 cc of a 1 per cent aqueous solution of *bichloride of mercury* until the condition is improved Then the same dose is given once weekly for maintenance This procedure is too new and unconfirmed to judge of its long range effectiveness

Miscellaneous—The injection of *liver extract* is said to be of value especially for improvement of the general state of health *Autohemotherapy* and large doses of *riboflavin* (100 mg daily) have not been of particular benefit *Moccasin venom* injections have been said to bring about remission but general confirmation of this is not at hand

Local Treatment—Local treatment of the skin is important A valuable procedure is *general body baths* in *warm potassium permanganate solution* (1:10,000) if the patient's condition allows them Local application of 1 per cent *gentian violet* in aqueous solution or 2 per cent *tannic acid solution* are helpful in overcoming the raw eroded condition of the skin Alkaline mouth washes are necessary for the oral lesions

PITYRIASIS ROSEA

Pityriasis rosea is a common acute and self limited disease characterized by a profuse eruption of *macular* pink scaling *ovate* patches on the trunk and extremities The cause of the condition is unknown Bacteria viruses and fungi have been held responsible Focal infection is said to play a part in some instances and the wearing of new damp clothing has been blamed Familial and conjugal outbreaks are extremely unusual which would argue against an infectious or contagious agent It is most prevalent in the *spring* and *autumn* months The female sex shows a somewhat higher incidence It may appear at any age but predominates in the age period from ten to thirty years

Eruption—The generalized eruption is often but not always preceded by a solitary large reddish *initial lesion* This "herald or mother patch" is noted on the trunk or extremities from three to ten days before the disseminate eruption makes its appearance It is frequently mistaken for a ringworm of the body but fungi are not present and its true nature becomes apparent with the advent of the generalized eruption

The typical lesion of pityriasis rosea is a rounded or oval *maculopapule* faint pink to red and partially covered with a fine branny yellowish *scale* The *ovate* lesion arranges itself on the trunk so that its long axis parallels the lines of cleavage of the skin It varies from 1 to 4 cm in length There may be a sparse eruption or lesions may be present in such profusion that they tend to coalesce and form large plaques (Fig 1000)

The usual lesion is dry but at times there is a distinct *vesicular element* In some instances too there is an exaggeration of the papular element bringing about considerably more elevation than is the rule When the rash disappears there is no residuum except occasionally for mild *leukoderma* The latter is especially true when the skin is exposed to ultraviolet light and is probably of similar mechanism to the *achromia* seen in *tinea versicolor* (p 3300)

The typical *distribution* of the eruption is on the *trunk neck arms* and *thighs*. There may be a few isolated patches below the elbows and knees. The trunk generally reveals the most abundant outbreak. Atypical distribution is not uncommon the lesions appearing on the face the palms the soles the genitals the scalp and even on the buccal mucosa. In persons wearing a bathing suit and exposed to the sun the eruption may involve only the areas covered by the bathing suit.

In the overwhelming majority of cases there are no *prodromes* and no *systemic symptoms*. There may be no subjective symptoms but mild to severe *itching* is often present. Frequently the itching is brought on



Fig 999 —Pemphigus



Fig 1000 —Pityriasis rosea. Note herald patch in picture at right

and aggravated by the excessive use of soap and water and the application of topical remedies. *Ultraviolet light* which is an effective treatment procedure may induce pruritus.

Course—The disease is self limited usually clearing spontaneously in four to eight weeks but occasionally persisting for three to four months. *Recurrences* are very rare but have been reported by competent observers.

Diagnosis—A pityriasis rosea like eruption may be elicited by sensitivity to *drugs* especially *arsphenamines* (p 116) and *gold salts* (p 2922). Coming on in the course of therapy such an eruption may lead to considerable perplexity as to whether there is a coincidental pityriasis rosea or a drug eruption having a similar morphology. The wisest and safest

procedure is to cease the administration of the drug since its further exhibition may bring on a generalized *exfoliative dermatitis* of serious significance. Observation may then show that the eruption pursues a course like that of ordinary *pityriasis rosea*. When it has cleared the drug may be resumed giving extremely small doses at first and increasing the amount slowly and gradually provided that there is no recrudescence of the rash.

Pityriasis rosea may need to be differentiated from secondary syphilis, *seborrheic dermatitis* and ringworm of the body. The *macular syphiloderm* never scales; there are concomitant evidences of syphilis and the *serologic tests* are positive. The *papular syphiloderm* is more elevated, darker in color and palpation reveals a deep infiltration of the lesions. *Seborrheic dermatitis* is a more chronic process, slower in its evolution and tends to be distributed more narrowly over the midline of back, presternal region, face and scalp. The scale is often yellowish and greasy. *Ringworm of the body* (p. 3293) rarely shows so great a profusion of lesions; the individual lesion tends to clear centrally, has a vesicular border and fungi are usually demonstrable with ease.

Treatment—Because the disease is self limited, *treatment is unnecessary*. Overenthusiastic therapy may lead to much suffering for the patient, bringing on widespread dermatitis with itching. The application of a simple *dusting powder* (p. 3137) or one medicated with 0.5 to 1 per cent *menthol* may be entirely adequate. *Starch baths* can be used but *soap and strong ointments* are undesirable.

There is general conviction that irradiation with *ultraviolet* or *cold quartz light* shortens the course of the disease. *Suberythema* doses should be used.

Recently success has been claimed by the injection of *serum* taken from patients convalescing from *pityriasis rosea*. This procedure is experimental and is not recommended for general usage. Vaccines prepared from *Schizosaccharomyces hominis* (Bendek) have been claimed to shorten the course appreciably. Those (Gougerot) who believe in the fungous causation, holding *Cryptococcus duboisii* at fault, have reported success with the application of 2 per cent eosin in 80 per cent alcohol.

PITYRIASIS RUBRA PILARIS

Pityriasis rubra pilaris is a chronic inflammatory skin disease characterized by an eruption of small, horny follicular *papules* which may become very extensive and eventuate in *exfoliative dermatitis* (p. 3383). The condition is rare. The cause of the condition is unknown. Recent investigation suggests that *deficiency of vitamin A* is concerned. There is a close similarity of the eruption to the *follicular hyperkeratoses* observed in *vitamin A deficiency* (p. 619). A study of three patients reported in the recent literature showed definite evidences of impaired dark adaptation (night blindness) and coincidental improvement in this as well as the skin lesions after therapy with *vitamin A*.

The disease may begin in the adolescent period or in later life, between forty and sixty years. Males have a higher incidence than females.

Eruption—The typical *papule* is fine, pointed, firm and dry and is situated at the mouth of the *hair follicle*. It is pink or red or, at times, the color of the normal skin. Each *papule* is penetrated by a *hair* and topped by a *horny deposit* which surrounds the hair and dips into the follicle.

The eruption may appear on any portion of the skin often attacking the *face neck or extremities* at first. On the dorsum of the proximal and middle phalanges of the *digits* the papules produce a very characteristic appearance. The hairs break off flush with the skin and the papules then seem to have a *black central core*. The papules are arranged in groups covering large or small areas and may involve almost the entire skin surface. When the hand is rubbed over these areas there is a sensation of roughness which has been aptly compared to that of a nutmeg grater. In addition to the papular eruption there are usually present *profuse scaling and crusting of the scalp a keratodermic thickening of the palms and soles and dystrophy of the nails*.

Pathology—The histopathology reveals a characteristic *hyperkeratosis* horny plugs within dilated follicles some acanthosis and mild inflammatory reaction in the upper cutis.

Course—The disorder commences insidiously and progresses slowly. It may be localized generalized or in rare instances universal. When widespread with coalescence of the papules and much scaling it may produce the picture of exfoliative dermatitis. There are few or no subjective symptoms and the general health is usually unimpaired. The disturbance may go on for many years but spontaneous remissions do take place with or without subsequent relapse.

Diagnosis—When the condition is fully developed with the typical follicular papules the black cored papules on the dorsum of the phalanges and the hyperkeratosis of the palms and soles it is difficult to confuse with any other skin disease. It lacks the scale and distribution of *psoriasis* (p 3414) the characteristic violaceous papules of *lichen planus* (p 3389) and it is always more widespread than *leratosis pilaris* (p 3236). When it assumes the appearance of an *exfoliative dermatitis* (p 3383) biopsy may be necessary for diagnosis.

Treatment—The most hopeful therapy is the administration of *vitamin A* in high dosage (100 000 to 200 000 units daily). The skin may be treated with an ointment containing 2 per cent *salicylic acid* in equal parts of *lanolin* and *petrolatum*. Since vitamin disturbances are often multiple the patient should be given a full diet enriched with vitamins. *Liver extract injections* and *Fowler's solution* are also recommended.

POIKILODERMA ATROPHICANS VASCULARE

Poikiloderma atrophicans vasculare is a rare cutaneous disorder resembling chronic radiodermatitis. Its existence as a primary entity is still under question. It has been observed chiefly in adults and in a number of instances it has been the precursor of *granuloma fungoides* (p 3386). At times a similar if not identical condition is observed in association with *dermatomyositis scleroderma* and *acute disseminated lupus erythematosus*. Some consider it a *secondary manifestation of injury of the small cutaneous blood vessels brought on by a variety of noxious agents*.

The Eruption—The disease is slow and insidious in its onset and progress and may remain active for many years. The sites of predilection are the *flexural regions* though the condition may be very widespread. The face is generally spared but involvement of the eyelids seems not uncommon.

The typical changes are reticulated brown *pigmentation lichenoid pap*

procedure is to cease the administration of the drug since its further exhibition may bring on a generalized *exfoliative dermatitis* of serious significance. Observation may then show that the eruption pursues a course like that of ordinary *psoriasis*. When it has cleared the drug may be resumed giving extremely small doses at first and increasing the amount slowly and gradually provided that there is no recrudescence of the rash.

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Treatment—Because the disease is self limited, treatment is unnecessary. Overenthusiastic therapy may lead to much suffering for the patient, bringing on widespread dermatitis with itching. The application of a simple *dusting powder* (p. 3137) or one medicated with 0.5 to 1 per cent *menthol* may be entirely adequate. *Starch baths* can be used but *soap* and *strong ointments* are undesirable.

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 DIFFERENTIAL DIAGNOSIS OF

Pruritus Ani

See also Generalized Pruritus p 3170 Localized Pruritus p 3173 Pruritus Vulvae p 2594
 In addition to the causes which produce generalized pruritus (p 3170) the under noted local conditions often cause the maddening sensation of it hung in the region of the anus. Eventually whatever the initial cause of the symptom secondary complications are superimposed in the nature of excoriations intertrigo dermatophytosis or lichenification

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|-----------------------|---|
| Allergy | Without local lesion. Often due to hypersensitivity to digestant or cathartic. Try elimination tests (p 552) |
| Constipation | Usually of rectal type with impaction of feces |
| Contact dermatitis | From clothes toilet paper sanitary napkins douches or suppositories. Get history. Make patch test |
| Dermatophytosis | Superficial fungous infection. Usually secondary to excoriation or continued moisture. Isolate pathogen. Consider x ray therapy (p 3293) |
| Diarrhea | From whatever cause (p 1840). Particularly after saline catharsis |
| Hemorrhoids | Continued moisture of opposing surfaces produces itching and invites secondary dermatophytosis (p 1916) |
| Helminthiasis | Particularly oxyuriasis and ascariasis. Look for ova and parasites in stool. Identify locally by method using scotch tape (p 1833) |
| Hygienic error | Uncleanliness due to carelessness in local cleansing |
| Intertrigo | Usually secondary to errors in hygiene dermatophytoses or hemorrhoids (p 3161) |
| Incontinence of feces | Due to prolapse of rectal mucosa or fistula. Secondary intertrigo and dermatophytosis. Correct initial lesion (p 1915) |
| Psychogenic | Of unusual frequency. May be complicated by secondary excoriation lichenification or dermatophytosis. May respond to tattooing. Consider reference to psychiatrist (p 1344) |
| Pediculosis pubis | Look for nits lice and crabs. Examine local hair and clothing |
| Polycythemia | Frequently associated with lichenoid dermatosis producing intense pruritus. Get hemogram (p 1092) |
| Psoriasis | Frequently associated with local lesion complicated by secondary lichenification or dermatophytosis. Look for lesions elsewhere (p 3414) |
| Vulvovaginal | Resulting from irritating discharges from vulva or vagina (p 2586) |

The *acute* form is rather widely distributed usually being most profuse upon the *trunk*. More often the eruption develops in a gradual manner a

ules with slight scaling and closely set profuse telangiectases giving the skin a marbled, bright red appearance Atrophy of the skin invariably occurs with the passage of time Unlike dermatomyositis edema is generally absent and the characteristic myositis muscular pain and weakness are also lacking

Diagnosis and Treatment—The condition must be differentiated from chronic radiodermatitis and chronic actinic dermatitis on the basis of history and histologic changes Dermatomyositis acute disseminated lupus erythematosus and scleroderma can usually be ruled out by study and observation

There is no satisfactory therapy The disease may develop into a lymphoblastoma

PSORIASIS

Psoriasis is a chronic inflammatory disease of unknown causation characterized by the appearance of scaly papules and patches It is a capricious disease both in appearance and course but it tends to assume certain clearly recognizable patterns These will be described with the understanding that variations and admixtures are frequent

Etiology—The cause of psoriasis is not known The disease may begin at any period from infancy to old age but most commonly appears some time between adolescence and the thirtieth year Statistical surveys show an average familial incidence of 25 per cent suggesting that hereditary influences may be involved It occurs about equally in the sexes

There is no satisfactory evidence to support the beliefs that psoriasis is due to infection by any known type of micro-organism to organic or functional derangements of the nervous system to a deficiency of vitamins or to endocrine gland dysfunction A variety of clinical and experimental observations have led to the proposal of these theories but none has stood the test of time

The concept that a metabolic disturbance underlies psoriasis was furthered by studies which showed a disturbance of the nitrogen metabolism leading to the recommendation of a low protein diet However there has been no adequate substantiation of these findings A more practical theory that holds promise for therapeutic relief deals with the disturbances of lipid metabolism The blood lipids are usually elevated and it has been thought that the condition might be due to an inability properly to metabolize fat Treatment with low fat or fat free diets has had considerable success in some instances but there has been no consistency about the favorable results The isolation from the pancreas of an internal secretory substance named lipocain which is concerned with the metabolism of fat gives promise of clarifying the etiology of psoriasis and holds considerable therapeutic promise

Pathology—The essential changes in psoriasis are parakeratosis acanthosis and elongation and narrowing of the papillae The stratum granulosum is thin or even absent Munro abscesses (micro abscesses) are small circumscribed accumulations of leukocytes in the stratum corneum In pustular psoriasis the Munro abscesses are unusually large The blood vessels of the upper cutis are dilated and there is a sparse infiltration of round cells

Clinical Manifestations—Psoriasis may appear in a variety of forms relative to the eruption and its site

Psoriasis Vulgaris—The common form of psoriasis (psoriasis vulgaris) may appear more or less acutely as a disseminated eruption of small red spots which soon enlarge and become mildly papular This is the so-called acute or eruptive form of the disease It is here that the Koebner phenomenon is often present as in lichen planus (p 3389) and dermatitis venenata (p 3330) This refers to the development of papules in scratch marks or in any normal area which is subjected to trauma There may be only a fine scant scale or the typical silvery white scale may develop

patchy or diffuse. The patchy type consists of variously sized plaques covered with a whitish scale and set off sharply from the healthy scalp. At times the scale is somewhat yellowish in color. The diffuse type may cover the entire scalp with a cap of grayish white or silvery white scale, removal of which reveals a reddened base. In some cases the scalp lesions spread by contiguity to the adjacent skin. Usually too there is no loss of hair.

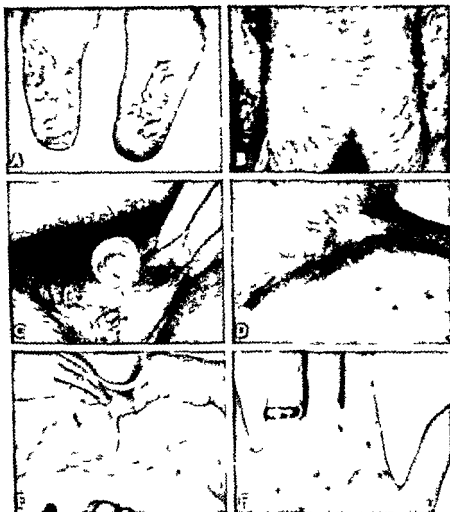


Fig. 1001.—Psoriasis. A Heels B Generalized C Penis D Anus E Under breasts F Parapsoriasis variant formis

Psoriasis of the Nails—Psoriatic changes of the nails often accompany the cutaneous eruption but they may be absent and in unusual instances they may exist alone. There are two distinct varieties. The stippled or thimble nail reveals a few or many tiny rounded pits. They have been likened to the appearance of the side of a thimble. Generally several nails or all of them show a variable number of these pits. The second form

few lesions appearing at a time and the condition gradually progressing until it reaches its maximal extent. The *papule* or *patch* is sharply delimited from the normal skin slightly elevated minimally infiltrated dry and covered with the characteristic laminated silvery white *scale*. The scale is readily scraped away disclosing the reddened base and a number of discrete tiny *bleeding points*. These are the result of the exposure and rupture of the capillaries of the papillae which are abnormally close to the skin surface in psoriasis. This phenomenon is of some diagnostic value and is called the *Auspitz sign*.

The lesions may vary from the size of a pinhead to areas larger than a man's hand. They are frequently called by descriptive terms according to their size. Thus *punctate psoriasis* refers to lesions about the size of a pin head, *guttate psoriasis* to those the size of a drop and *nummular psoriasis* to those the size of a coin. They tend to be oval or rounded in outline but may assume irregular shapes. Sometimes as they enlarge peripherally they clear centrally and then have an *annular* configuration. Also as they extend the lesions may coalesce and produce a great variety of bizarre shapes. In the individual patient they may be more or less uniform or there may be a great disparity in size and shape. Only one or two lesions may be present or there may be dozens.

Psoriasis of the common variety usually distributes itself in a remarkably consistent fashion. The sites of predilection are the *extensor surfaces of the elbows and forearms*, the *knees*, *legs*, *scalp* and *sacrum*. In a given patient all these areas may be involved at the same time but this is not necessarily so. However there is a distinct tendency to *bilateral symmetrical distribution*. Variations are not uncommon and may give rise to considerable difficulty in diagnosis.

Inverse Psoriasis—Inverse psoriasis attacks the *flexor* rather than the extensor surfaces particularly the *axillae*, *submammary folds*, *antecubital fossae*, *groins*, *perineum* and *intergluteal folds*. The eruption consists of diffuse reddened slightly thickened *patches*. The discrete typical psoriatic papule may not be present. Scaling is often scanty or absent and the area may be somewhat moist and pruritus well marked. At first glance there is a resemblance to *dermatitis intertrigo* (p 3161) or *monilial infection* (p 3301).

Universal Psoriasis (Psoriatic Erythroderma)—Universal or generalized psoriasis is rare and usually a sequence of the common form. The so-called acute variety is especially prone to become generalized if the skin is treated harshly. *Strong ointments*, *ultraviolet light* and *arsenic* should not be used in the acute eruptive form lest they lead to universal dissemination of the eruption. The transition may however take place spontaneously. Universal psoriasis assumes the appearance of *dermatitis exfoliativa* (p 3383) and is often associated with *arthritis* (p 3418).

Inveterate Psoriasis—Inveterate psoriasis usually has the typical morphology of the disease except that it is more often *infiltrated*. There are as a rule only a few patches present. They are distinguished by their lack of response to therapy and their chronicity.

Psoriasis of the Scalp—Psoriasis may confine itself to the scalp the skin and nails being completely free. Usually however there are concomitant lesions on the glabrous skin. In the scalp the eruption may be

forms. It is particularly characteristic of the inverse variety and also where the eruption is eczematized or generalized. Secondary eczematous changes in a psoriatic patch may be due to the trauma of scratching to the application of irritating medicaments and to friction sweating and maceration chiefly in the flexural regions.

The pustular and universal varieties are very refractory to treatment. It is most difficult to ameliorate psoriatic affection of the nails.

Differential Diagnosis—The main diagnostic features of psoriasis are (1) its characteristic *distribution* on the *extensor surfaces* of the extremities and on the scalp (2) the reddish dry slightly elevated but not infiltrated sharply demarcated *papules* covered with a *silvery white scale* (3) the typical changes in the *nails* (4) the *pinpoint bleeding* when the papule is scraped (5) the *chronicity* of the disease (6) the tendency to *familial occurrence* in 25 per cent of patients and (7) the distinctive *histopathology*.

The common form of psoriasis must be differentiated from *seborrheic dermatitis* (p 3432) the papulosquamous secondary *syphiloderm* (p 3281) *pityriasis rosea* (p 3410) *localized neurodermatitis* (p 3279) and *hypertrophic lichen planus* (p 3389). Pustular psoriasis may be confused with *fungous conditions* (p 3293) and *dermatitis venenata* (contact dermatitis) (p 3330). Contact dermatitis can usually be ruled out readily by study and observation by removal of the patient from the probable source of irritation and by patch tests. Dermatophytids of the hands are generally accompanied by fungous lesions of the feet and a positive trichophyton test (p 60) as a rule they have a somewhat different appearance and distribution.

Treatment—There is no one satisfactory treatment for psoriasis but by proper management most eruptions can be controlled at least temporarily. Local therapy is usually most satisfactory whereas the list of systemic remedies by its very length casts doubt on their efficacy.

Local Treatment—In the acute or eruptive phase of psoriasis local treatment should be of the blandest variety. *Boric acid ointment* or *petrolatum* is used on the skin and *starch baths* are indicated if there is itching. Scales are removed by the bath which may be plain or medicated with bicarbonate of soda or starch. After the removal of the scales the prescribed ointment is rubbed in thoroughly.

The following prescriptions may be used in the *milder types*

| | |
|-----------------------|------------|
| R. Salicylic Acid | 1.5 |
| Lanolin | |
| Petrolatum | ad 60.0 |
| I. Salicylic Acid | 1.5 |
| Ammoniated Mercury | 3.0 |
| Petrolatum | qs ad 60.0 |
| R. Salicylic Acid | 1.5 |
| Ol of Cade | 3.0 |
| Petrolatum | qs ad 60.0 |

When the lesions are *thicker* and more *infiltrated* they require reducing agents of *greater strength*. The following prescriptions may be used

consists of a *subungual papule* usually beginning at the lateral margin and appearing as a yellowish thickening with *subungual hyperkeratosis*. As the condition progresses the nail is raised and deformed and takes on an opaque yellowish lusterless appearance. In this type as a rule only a few nails are involved. At times the adjacent skin of the fingertips is affected and shows reddened scaling patches.

Psoriasis of Face Palms Soles and Genitals—Psoriasis does not commonly appear on the face, the palms, soles or genitals. When these areas are affected alone diagnosis is difficult but when typical lesions are present elsewhere the interpretation is fairly simple. On the *face* there are discrete papules or plaques with more or less characteristic scale. On the *palms and soles* the lesions are at first diffuse pink slightly infiltrated and scaly patches. Later there is a tendency to considerable hyperkeratosis and fissuring. *Genital psoriasis* consists of superficial slightly scaly reddened patches on the glans or shaft of the penis or on the labia majora. When these are the sole lesions present the diagnosis is anything but easy.

Pustular Psoriasis—Pustular psoriasis is a rare variant characterized by its *acute* manner of onset, its protracted course and its localization on the *palms and soles*. Typical psoriatic lesions may or may not be present elsewhere. When they are absent the diagnosis becomes very difficult. The eruption consists of more or less profuse tiny whitish *pustules* which rupture and become converted into reddened *scaly patches*. There is a tendency for new crops of pustules to erupt while the older ones are involuting. Subjective symptoms are common and include itching, burning and stinging sensations. The histopathology is strongly suggestive of psoriasis except that the micro abscesses are exceptionally large.

Arthropathic Psoriasis—Arthropathic psoriasis is a rare type in which arthritis and psoriasis coexist. The articular manifestations commence typically in the *fingers*, spreading slowly to involve the *larger joints* of the extremities and the *spinal column*. When the skin lesions become aggravated there is usually an exacerbation of the arthritis. This fact among others suggests that the two conditions are related. The skin eruption may assume the character of any form of psoriasis but generalized or universal types are not uncommon.

Course—The common form of psoriasis may pursue a more or less acute course but essentially it is a *chronic disease*. The acute variety especially may disappear in a comparatively short period or it may take on characteristics of the chronic form.

Psoriasis may persist for many years with little or no change. On the other hand there may be long periods of *remission* followed by *relapse*. The eruption may disappear with or without the benefit of therapy and never recur and it is this marked capriciousness of the disease which makes it difficult to evaluate the effectiveness of any therapy. Even though the patient cannot be promised a cure treatment offers the opportunity of months or even years of freedom from the eruption.

When the psoriatic papule disappears there is rarely a residuum. At times it may be followed by a transient white spot of *depigmentation* (psoriatic leukoderma) or by a brown *hyperpigmented macule*. These pigmentary abnormalities disappear gradually in a variable period of time. Psoriasis is not an itching dermatosis but there are many exceptions to this rule. *Pruritus* may be complained of bitterly in any of the various

In the morning the skin is cleaned with olive oil and a thin film left in place. The body is then exposed to *ultraviolet light* starting with small dosages and gradually increasing the length of exposure.

For the *pustular eruptions* hot soaks of *potassium permanganate* (1:3000 to 1:10,000) are used during the daytime every two hours for about fifteen minutes. At night *ammoniated mercury ointment* 3 to 5 per cent or *chrysarobin* in chloroform 1 to 3 per cent is applied. The hands should be protected with white cotton gloves during the night.

Heliotherapy which often proves most efficacious may be obtained by exposure to *natural sunlight* or to the *ultraviolet light*. In the opinion of most observers natural sunlight seems to be more beneficial.

Radiotherapy is used only in the eczematized variety and in cases where there exist one or two isolated inveterate patches. It should not be used in the common variety since the disease has a natural tendency to recur. The response to the roentgen ray may encourage the patient to keep seeking this modality and there is then the danger of chronic radio dermatitis. It has been noted too that psoriatic patches which have once been cleared with the roentgen ray are more intractable to topical treatment when they recur.

Systemic Therapy—Systemic therapy is usually employed in psoriasis. There is considerable difference of opinion as to its value and the multiplicity of the remedies that are suggested implies that no one is of any specific value. As usual arsenic is given as Fowler's solution (p. 116) or by injection as sodium arsenate or Enesol. Massive doses of vitamin D 200,000 to 500,000 units daily have been reported to be of benefit. Vitamin B complex and injections of liver extract are also employed as are injections of colloidal manganese 1 cc weekly. Autohemotherapy using intramuscular injections of 5 to 10 cc of the blood drawn from the patient's vein and injections of a foreign protein such as boiled milk or typhoid vaccine have been suggested.

Dietary measures have also many advocates. Low protein and low fat regimens have been widely used with disappointing results. The most recent metabolic contribution has been the oral administration of defatted wheat germ 30 to 45 gm daily or soy bean lecithin 30 to 60 grams daily. Encouraging results have been reported from these preparations. The further experiences with lipocaine are being watched with the greatest hope and a specific may finally be discovered.

PARAPSORIASIS

Parapsoriasis is a term applied to a group of rare resistant scaly eruptions marked by their *chronicity* and lack of subjective symptoms. The cause of parapsoriasis is unknown. Except for the varioliform type it is seen chiefly in later adult life. Its chief significance is the possibility of confusion with the prefungoid eruptions of *granuloma fungoides*.

Clinical Manifestations—Several distinct varieties of parapsoriasis are described. *Guttate (lichenoid) parapsoriasis* consists of a generalized non-itching eruption of small flat papules of a reddish hue covered with a fine scale. At times it closely resembles psoriasis in size and scaling but the histopathology is different. It must also be differentiated from the papular syphiloderm but the chronicity, lack of infiltration, absence of concomitant

| | |
|--------------------|------------|
| ℞ Salicylic Acid | 2.0 |
| Ammoniated Mercury | 60 |
| Petrolatum | q.s ad 600 |
| ℞ Salicylic Acid | 1.5 |
| Oil of Cade | 30 |
| Ammoniated Mercury | 30 |
| Petrolatum | q.s ad 600 |

In the *resistant severest types* of eruption the most satisfactory remedies are *chrysarobin* or *anthralin* (dihydroxvanthranol) These may be used in the following manner

| | |
|----------------------|------------------|
| ℞ Chrysarobin | 0.6-2.0 |
| Lanolin | |
| Petrolatum | aa q.s ad 600 |
| ℞ Salicylic Acid | 1.5 |
| Chrysarobin | 1.2 |
| Lanolin | |
| Petrolatum | aa q.s ad 600 |
| ℞ Chrysarobin | 10-30 |
| Chloroform | q.s ad 300 |
| ℞ Crude Coal Tar | 30 |
| Petrolatum | q.s ad 600 |
| ℞ Anthralin Ointment | 0.1-0.5 per cent |
| ℞ Chrysarobin | 60-120 |
| Oil of Cade | 60-120 |
| Salicylic Acid | 30-60 |
| Soft Soap | |
| Petrolatum | q.s ad aa 1000 |

(This is a modification of Drews ointment and is particularly useful for inveterate patches)

When the psoriatic patches are *eczematized* and moist it is best to use blander applications Of value are 1 per cent aqueous solution of *Gentian Violet* 1 to 3 per cent solution of *Silver Nitrate* or the lotion of *Calamine and Zinc* containing 3 per cent of *Liquor Carbonis Detergens* (p 3131)

For lesions on the *face* weaker ointments are preferable *Ammoniated Mercury Ointment* of 2 to 5 per cent strength or *Salicylic Acid Ointment* 1 to 3 per cent may be used *Chrysarobin* should never be applied to the face or scalp It dirties the hair and more importantly it may get in the eyes where it causes severe conjunctivitis and even keratitis In this region ammoniated mercury ointment is generally used and to it may be added salicylic acid and oil of cade In some cases sulfur is an excellent remedy but it should never be used immediately after or along with mercury An ointment of 2 to 5 per cent sulfur and 1 to 3 per cent salicylic acid may be prescribed

In cases where the eruption is generalized or universal the treatment developed by Goeckerman and O'Leary has proved of the greatest value At night before retiring a preparation of 2 per cent *coal tar* in ointment of *zinc oxide* is rubbed into the skin and allowed to remain overnight

| | |
|---------------------------------|---|
| Subacute bacterial endocarditis | Bacteremia petechiae and splinter hemorrhages under nails Get blood culture Start intensive treatment with penicillin and heparin (p 286 1050) |
| Typhus fever | Rickettsial infection with centrifugally advancing macular eruption May become hemorrhagic in severe infections Get Weil-Felix reaction (p 369) |
| Vaccinia | Widespread eruption following therapeutic or prophylactic vaccination May be hemorrhagic in severe instances (p 428) |
| Vitamin K deficiency | Hypothrombinemia resulting in diffuse hemorrhagic manifestations Note therapeutic response to menadione (p 1111) |
| Yellow fever | Severe virus infection with jaundice and hemorrhagic manifestations Evidences of hepatic and renal damage (p 477) |

evidences of syphilis and negative serologic tests of the blood usually suffice to clarify the syndrome (Fig 1001 F)

Variegated (retiform) parapsoriasis is probably a form of guttate parapsoriasis in which the lesions are very profuse and are so arranged that they produce a lacelike pattern Examples have been reported in which granuloma fungoides (p 3386) later developed but it is more likely that these were not originally parapsoriasis but the prefungoid stage of granuloma fungoides

Parapsoriasis en plaques is a rare form characterized by widely dispersed well demarcated superficial noninfiltrated pink to tan patches covered with a fine adherent scale They are intractable to therapy and subjective symptoms are absent This variety has also been said to be a precursor of granuloma fungoides It is most probable that those examples which later developed granuloma fungoides were not true parapsoriasis en plaques but were indifferent scaling patches which are often seen in the prefungoid stage of that disease

Parapsoriasis varioliformis (pityriasis lichenoides et varioliformis acuta) differs considerably from the other members of the group and may not even belong with them It occurs in younger persons has a comparatively acute course and its morphology is distinctly different It may be accompanied by mild constitutional symptoms The eruption consists of lichenoid papules as described in guttate parapsoriasis and papulovesicles suggestive of chickenpox or smallpox When the eruption clears there are often residual varioliform scars and leukodermic areas

Treatment—There is no effective therapy Inorganic arsenic vitamins roentgen rays and ultraviolet light have been recommended

PURPURA

Bleeding into the skin commonly occurs as a result of recognizable abnormalities such as trauma septicemia drug idiosyncrasy poisoning avitaminosis or blood dyscrasias such as hemophilia leukemia and the like

DIFFERENTIAL DIAGNOSIS OF

The Purpuric Lesions

See also Telangiectases p 3394 Purpuras p 3423

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|--|---|
| Anaphylactoid purpura | Eruption associated with abdominal pain and arthropathy Identify offending allergen (p 3424) |
| Drug eruptions | Dermatitis medicamentosa due especially to sulfonamides barbiturates gold, arsenic, iodide bromide and silver Check history |
| Endocrinologic | Menstrual and menopausal bruising |
| Erythema nodosum | Firm blue or red nodules on extensor surfaces of leg Associated with hypersensitivity usually to bacterial protein (p 3377) |
| Idiopathic thrombocytopenic purpura | Hemorrhagic tendency with low platelet count Consider splenectomy (p 1116) |
| Idiopathic multiple hemorrhagic sarcomas | Kaposi's disease Purplish nodular lesions of legs due to malignant dermatosis Get biopsy (p 3226) |
| Haverhill fever | Rat bite followed by generalized maculopapular eruption Purpuric lesions in severe instances (p 363) Give penicillin |
| Hemophilia | Congenital bleeding tendency of males Get history (p 1118) |
| Leukemia | Various dermatoses including purpuras in fatal disease of the blood forming organs Check hemogram and marrow smears (p 1100) |
| Measles | Acute exanthem with maculopapular eruption Purpuric lesions in malignant infections (p 409) |
| Meningococcemia | Bacteremia with petechiae purpuras and herpetic vesicles Check blood culture and cerebrospinal fluid (p 211) Start intensive treatment with penicillin parenterally and intrathecally |
| Rheumatic fever | Generalized infection with carditis and arthralgias Associated often with erythemas and purpuric nodules (p 186) Note response to salicylates |
| Rocky Mountain spotted fever | Rickettsial infection with centripetally advancing macular eruption May become hemorrhagic in severe infections (p 376) Get Weil-Felix reaction |
| Scurvy | Diffuse hemorrhagic manifestations due to deficiency of vitamin C Note therapeutic response to ascorbic acid (p 2857) |
| Smallpox | Acute exanthem with monomorphic papular vesicular and pustular lesions becoming hemorrhagic in severe infections (p 424) Try antibiotic treatment |
| Streptococcemia | Bacteremia with erythemas petechiae or purpuras Get blood culture Treat with penicillin (p 163) |

| | |
|---------------------------------|---|
| Subacute bacterial endocarditis | Bacteremia petechiae and splinter hemorrhages under nail. Get blood culture. Start intensive treatment with penicillin and heparin (p. 286, 1050). |
| Typhus fever | Rickettsial infection with centrifugally advancing macular eruption. May become hemorrhagic in severe infections. Get Weil-Felix reaction (p. 369). |
| Vaccinia | Widespread eruption following therapeutic or prophylactic vaccination. May be hemorrhagic in severe instances (p. 428). |
| Vitamin K deficiency | Hypothrombinemia resulting in diffuse hemorrhagic manifestations. Note therapeutic response to menadiolone (p. 1111). |
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evidences of syphilis and negative serologic tests of the blood usually suffice to clarify the syndrome (Fig. 1001 F).

Variegated (retiform) parapsoriasis is probably a form of guttate parapsoriasis in which the lesions are very profuse and are so arranged that they produce a lacelike pattern. Examples have been reported in which granuloma fungoides (p. 3386) later developed but it is more likely that these were not originally parapsoriasis but the prefungoid stage of granuloma fungoides.

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Parapsoriasis varioliformis (pityriasis lichenoides et varioliformis acuta) differs considerably from the other members of the group and may not even belong with them. It occurs in younger persons, has a comparatively acute course and its morphology is distinctly different. It may be accompanied by mild constitutional symptoms. The eruption consists of lichenoid papules as described in guttate parapsoriasis and papulovesicles suggestive of chickenpox or smallpox. When the eruption clears there are often residual varioliform scars and leukodermic areas.

Treatment—There is no effective therapy. Inorganic arsenic, vitamins, roentgen rays and ultraviolet light have been recommended.

PURPURA

Bleeding into the skin commonly occurs as a result of recognizable abnormalities such as trauma, septicemia, drug idiosyncrasy, poisoning, avitaminosis or blood dyscrasias such as hemophilia, leukemia and the like.

The present discussion refers to *idiopathic purpuras* whose dominant clinical manifestation is the eruption. These syndromes include

- Anaphylactoid Purpura (Hemorrhagic Capillary Toxicosis)
- Purpura Simplex
- Purpura Rheumatica (Schonlein or Peliosis Rheumatica)
- Henoch's Purpura
- Idiopathic Thrombocytopenic Purpura (Purpura Hemorrhagica)

Anaphylactoid Purpura (Hemorrhagic Capillary Toxicosis)—Anaphylactoid purpura includes purpura simplex, purpura rheumatica and Henoch's purpura. It is characterized by the recurrent nature of the eruption, the comparative mildness of the condition and the frequent association of other dermatoses, joint swelling and visceral symptoms. In none of these is there found any disturbance of the blood.

The hemorrhagic capillary toxicoses occur most commonly in children and young adults. The Henoch's variety is most frequently observed in infants and young children. Purpura simplex occurs in older children and rheumatic purpura in adults twenty to thirty years of age.

Pathogenesis—The exact cause of these syndromes is unknown but they are believed to be *allergic* in nature, probably due to *food sensitization* or *focal infection*. There is apparently an *increased permeability* of the capillaries. When plasma alone transudes, the resultant lesion is *erythematous* or *urticarial*; but when whole blood seeps out, purpura appears. The purpuras are related to other diseases of the so-called erythema group such as erythema multiforme, erythema nodosum, angioneurotic edema and serum sickness.

Purpura Simplex—Purpura simplex is usually unattended by systemic manifestations although mild *fever*, *arthralgia* and *gastrointestinal symptoms* occasionally coexist. The purpuric eruption is most common on the *legs* and *feet* but may appear on the neck, trunk and upper extremities. There is no bleeding from the mucosal surfaces. The condition generally lasts two to four weeks and *disappears spontaneously* but *recurrences* are frequent.

Rheumatic Purpura (Schonlein or Peliosis Rheumatica)—Rheumatic purpura is usually accompanied by a *polymorphous eruption* which may be *urticarial*, *erythematous*, *erythema nodosum* like, *erythema multiforme* like, *petechial* or *ecchymotic*.

The *petechial eruption* is often most intense about the *affected joints* and may be restricted to these areas. The disease may begin with *fever* and *sore throat* followed by painful swelling of the large joints and the sudden appearance of *petechiae*. There is no involvement of the mucous membranes. The condition lasts up to four weeks and *disappears spontaneously* although recurrences are rather frequent. There may be an increase in the sedimentation rate.

Henoch's Purpura—Henoch's purpura may present any or all of the cutaneous manifestations of rheumatic purpura in varied combination. *Arthritis* may also be present. In occasional cases, however, cutaneous and arthritic abnormalities are absent and the characteristic *gastrointestinal symptoms* are present alone. These include *colicky abdominal pains*, *nausea*, *vomiting*, *diarrhea* and at times *melena*. *Intussusception* (p. 1876) has resulted from hemorrhage and edema of the intestinal wall.

In both rheumatic and Henoch's purpura there is at times renal involvement with the production of *albuminuria* and *hematuria* resembling an acute nephritis (p. 2374)

Diagnosis—The differential diagnosis of simple and rheumatic purpura is ordinarily easy. The absence of mucous membrane involvement and blood changes rules out *thrombocytopenic purpura*. Rheumatic fever is seldom accompanied by purpura and usually shows severe systemic symptoms. Henoch's purpura is apt to be mistaken for an acute surgical abdominal condition as appendicitis, volvulus, intussusception or Meckel's diverticulitis. This is especially true when skin lesions are not present.



Fig. 1002—Purpura simplex



Fig. 1003—Periorbital eczema of face

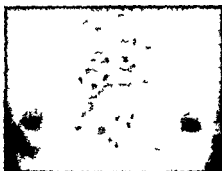


Fig. 1004—Periorbital dermatitis of chest

Treatment—Treatment is directed at improvement of the constitutional condition, the removal of possible food and drug allergens and the eradication of foci of infection. Ascorbic acid, salicylates, calcium and iron may be used.

Idiopathic Thrombocytopenic Purpura (Purpura Hemorrhagica)—This is a primary disease of unknown etiology. It is characterized by marked thrombocytopenia and bleeding from mucous membranes and into the skin. The condition is serious and is usually attended by more or less severe secondary anemia.

In addition to the thrombocytopenia and secondary anemia the blood

shows *prolonged bleeding time* and *poor clot retraction* The tourniquet test is positive indicating *increased capillary fragility*

The profuse hemorrhage from the nose mouth bowel or vagina overshadows the skin manifestations The latter may consist of a small number of *petechiae* or large areas of *ecchymosis* may be present

Blood transfusion followed by *splenectomy* usually leads to cure

SCLEREDEMA ADULTORUM

Scleredema adutorum is an uncommon disease characterized by diffuse induration of the deeper part of the cutis subcutaneous tissue fascia and muscles

Etiology—The cause is unknown but the condition is almost invariably preceded by an acute infection such as scarlet fever tonsillitis grippe influenza or measles It is believed that toxins elaborated in the course of the infection lead to deposition of a *mucinoid substance* in the affected tissues *Endocrine dysfunction* similar to the myxedema of hypothyroidism may be a factor It has also been suggested that this syndrome is not a disease entity but an unusual variety of *trophedema* due to nerve injury It may well be that *bacterial toxins* act upon the endocrine glands the involuntary nervous system or both to produce scleredema The condition is more frequent in the *female sex* and appears most often between the age of ten and forty years Instances in younger and older persons are however not rare

Clinical Manifestations—The manifestations come on within a few weeks after an acute infection with *swelling of the back of the neck* The patient may first complain of localized *pain* or *difficulty in flexing the neck* The swelling makes its way rapidly by direct continuity to the face shoulders arms and trunk Usually the hands and feet are not affected The swelling is hard almost boardlike painless and does not pit on pressure The brawny induration about the joints may impair articular movements

Course—In the average case there are rarely any systemic symptoms and *spontaneous recovery* ensues in six to eighteen months In some patients the lesion is of longer duration and there are also some in whom the induration never entirely disappears *Recurrences* are not uncommon The attacks may be separated by many years of complete normalcy

Diagnosis—The differential diagnosis of scleredema may present considerable difficulty especially from the early or edematous stage of diffuse scleroderma dermatomyositis and myxedema *Diffuse scleroderma* (p 3427) does not necessarily follow an infectious process It commences commonly in the hands and feet and its tempo of spread is far slower Pigmentation atrophy and the characteristic ivory tint of the skin are never present in scleredema adutorum *Dermatomyositis* (p 3373) is attended by muscular pain and tenderness fever erythema and other cutaneous manifestations and in over 50 per cent of cases causes death within the first two years *Myxedema* (p 1193) is a far more gradual process the nonpitting edema is most marked on the extremities there is increased weight decreased mental and physical activity alopecia and a lowering of the basal metabolic rate

Treatment—Treatment is empiric spontaneous involution being the rule

in a variable period of time Warm baths massage and diathermy are advocated Thyroid extract has been claimed to be of value

SCLERODERMA

Scleroderma is a rare chronic systemic disease characterized by localized or generalized induration of the skin The generalized or diffuse form is a serious and often fatal disease Scleroderma is far more frequent in females than males and may appear at any age from infancy to old age However the diffuse form is most frequent in adolescence and young adulthood the circumscribed type in older adults

Etiology—The cause of scleroderma is unknown The onset of the disorder after an acute infection such as tonsillitis scarlet fever measles or influenza suggests the possibility of a bacterial toxic origin Syphilis has been accused because it coexisted in a number of instances Nervous shocks and chronic intoxication with arsenic have also been proposed as etiologic agents The largest body of opinion inclines to the view that dysfunction of the endocrine and involuntary nervous systems are at fault and that the disease is in essence an angiotrophoneurosis Disturbances of the endocrines are not uncommon especially of the thyroid parathyroid and adrenal glands Vasomotor symptoms particularly those encountered in Raynaud's disease are frequently present It may be that a metabolic disturbance of calcium plays some part

Diffuse Scleroderma—Diffuse or generalized scleroderma may commence insidiously or the skin changes may be preceded by *malaise arthralgia loss of weight and weakness* In some patients the leading symptom is a typical Raynaud's syndrome (p 1000) The patient soon complains of *pain swelling and stiffness of the hands* less often of the face neck and trunk However as the disease progresses there is usually involvement of the entire skin surface intensification of systemic symptoms loss of weight weakness arthralgia and finally prostration and death The Raynaud like syndrome in the hands and feet sometimes dominates the symptoms

The earliest change in the skin consists of an *edemalike infiltration* which lacks the depth of scleredema adultorum (p 3426) The skin is thickened and indurated and cannot be pinched up but otherwise is normal or only slightly reddened With the advance of the infiltrative process the skin becomes harder whitish or ivory colored and bound down to the subjacent structures The rigidity of the skin interferes with normal function especially movement of joints the mouth and eyelids Involvement of the face leads to a masklike immobile mien The stage of infiltration and induration is followed by *atrophy* in which the skin becomes boardlike thinned and immovably adherent to the underlying tissues The ivory hue is altered by the advent of patchy or diffuse brown *pigmentation* Trophic ulcerations over bony points are not infrequent Telangiectasis is common and deposits of calcium sometimes appear in the subcutaneous tissues The hands and face take on the characteristics already described for *acrosclerosis* (p 3367) which many believe is a variant of scleroderma The face becomes thin expressionless cadaverous the nose pinched the mouth narrowed and the chin puckered The hands reveal the picture of *sclerodactylia* being semiflexed practically immobile the fingers rounded or tapered and the skin hardened and bound down firmly *Atrophy ulcers*

shows *prolonged bleeding time* and *poor clot retraction*. The tourniquet test is positive indicating *increased capillary fragility*.

The profuse hemorrhage from the nose, mouth, bowel or vagina overshadows the skin manifestations. The latter may consist of a small number of *petechiae* or large areas of *ecchymosis* may be present.

Blood transfusion followed by *splenectomy* usually leads to cure.

SCLERODEMA ADULTORUM

Scleredema adultorum is an uncommon disease characterized by diffuse induration of the deeper part of the cutis, subcutaneous tissue, fascia and muscles.

Etiology—The cause is unknown but the condition is almost invariably preceded by an acute infection such as scarlet fever, tonsillitis, grippe, influenza or measles. It is believed that toxins elaborated in the course of the infection lead to deposition of a *mucinoid substance* in the affected tissues. *Endocrine dysfunction* similar to the myxedema of hypothyroidism may be a factor. It has also been suggested that this syndrome is not a disease entity but an unusual variety of *trophoedema* due to nerve injury. It may well be that *bacterial toxins* act upon the endocrine glands, the involuntary nervous system or both to produce scleredema. The condition is more frequent in the *female sex* and appears most often between the age of ten and forty years. Instances in younger and older persons are however not rare.

Clinical Manifestations—The manifestations come on within a few weeks after an acute infection with *swelling of the back of the neck*. The patient may first complain of localized *pain* or *difficulty in flexing the neck*. The swelling makes its way rapidly by direct continuity to the face, shoulders, arms and trunk. Usually the hands and feet are not affected. The swelling is hard, almost boardlike, painless and does not pit on pressure. The brawny induration about the joints may impair articular movements.

Course—In the average case there are rarely any systemic symptoms and *spontaneous recovery* ensues in six to eighteen months. In some patients the lesion is of longer duration and there are also some in whom the induration never entirely disappears. *Recurrences* are not uncommon. The attacks may be separated by many years of complete normalcy.

Diagnosis—The differential diagnosis of scleredema may present considerable difficulty especially from the early or edematous stage of diffuse scleroderma, dermatomyositis and myxedema. *Diffuse scleroderma* (p. 3427) does not necessarily follow an infectious process. It commences commonly in the hands and feet and its tempo of spread is far slower. Pigmentation, atrophy and the characteristic ivory tint of the skin are never present in scleredema adultorum. *Dermatomyositis* (p. 3373) is attended by muscular pain and tenderness, fever, erythema and other cutaneous manifestations and in over 50 per cent of cases causes death within the first two years. *Myxedema* (p. 1193) is a far more gradual process; the nonpitting edema is most marked on the extremities; there is increased weight, decreased mental and physical activity, alopecia and a lowering of the basal metabolic rate.

Treatment—Treatment is empiric, spontaneous involution being the rule.

Course—Diffuse scleroderma is to a certain extent an unpredictable disease and may pursue a rapid course to a fatal termination in about one year or the patient may linger on for many years. Spontaneous arrest and remission occurs but rarely. In the average patient the disease progresses slowly for years and death finally results from intercurrent infection. In very exceptional instances actual retrogression of skin lesions may take place with considerable functional restoration.

Circumscribed Scleroderma—Circumscribed scleroderma is observed in several forms the most common being morphea and scleroderma in bands. Localized scleroderma is seen at times in conjunction with *progressive facial hemiatrophy* (p 2834).

Morphea is not associated with systemic symptoms and its prognosis is excellent recovery being the rule. It commences as a *reddish spot* about 1 to 8 cm in diameter which increases slowly in size. Later the center becomes *whitened* or *yellowed* while the periphery retains a violaceous hue. There is only slight thickening and in time complete *resolution* ensues leaving a thin atrophic slightly depressed white macule as a permanent stigma. Ordinarily only one lesion is present but several spots are not rare. The trunk and thighs are common sites of the lesions but other regions may be affected. The condition is more common in females in adult life.

The *linear* or *band form* of scleroderma has often appeared after *local injury*. It may be only a few inches long or may extend the length of an entire extremity. A characteristic localization is the *middle of the forehead* and *scalp* giving the so-called *saber cut* appearance. The lesion is an indurated whitened linear lesion which may be a fraction of an inch to several inches wide.

Diagnosis—The diagnosis of scleroderma is simple in the fully established case but in the early phases some difficulty may be encountered in distinguishing it from Raynaud's disease, chronic arthritis, dermatomyositis and scleredema adultorum.

Pathology—The chief pathological change is in the *collagenous tissues* which proliferate and become swollen. The blood vessels are secondarily affected becoming sclerotic and obliterated and thence leading to atrophy.

Treatment—The *localized forms* of scleroderma are self limited and involute spontaneously. Gentle massage with cocoa butter or lanolin is all that is required.

The *diffuse form* is a therapeutic problem which is far from solved. A nutritious diet enriched with vitamins is recommended as well as the administration of iron and arsenic (Fowler's solution). Improvement has been claimed from endocrine preparations particularly thyroid and pituitary extracts. Injections of an insulin free extract of pancreas have been warmly advocated. Vitamin D given in large doses (200 000 to 300 000 International Units daily) for at least four months has been said to bring about remission. Recently there has been reported excellent improvement after the prolonged oral administration of dihydrotachysterol (p 621). This drug as well as massive doses of vitamin D must be used cautiously because of their mobilizing action upon calcium depots. This may result in decalcification of the bones, renal lesions and nephrolithiasis.

Parathyroidectomy and ganglionectomy have been attended by only temporary relief.



Fig. 100 —Scleroderma (diffuse) Note the shrunken appearance of facies (Courtesy of Dr Paul Gross)



Fig. 100B —Sclerodactylia Note trophic ulcers at tips of fingers and claw like deformity of the hands (Courtesy of Dr A. M. Davidson, Winnipeg, Canada)

tions scars gangrene and spontaneous amputation of terminal phalanges may ensue

of large waxy yellowish scales which may mat together with the hair and form a thick dry crust Alopecia is frequently present

Treatment—The treatment of *oily seborrhea* must be directed at the scalp as well as the nonhairy surfaces The general health should be improved Fresh air sunshine regular exercise and a simple low fat diet are advised The addition of *liver yeast* and *vitamin B complex* to the diet is desirable Frequent washing of the skin with soap and water and for women a face powder containing sulfur are recommended Ordinary white soap may be used but in stubborn cases *Tincture of Green Soap* may be more satisfactory *Resorcin* and *sulfur* are effective remedies and may be prescribed as the *lotions* or *salves* which are used in *acne vulgaris* (p 3302) They should be used in low concentration at first as 1 per cent resorcin and 2 per cent sulfur and the strength increased if no irritation is produced *Salicylic acid* (1 to 2 per cent) and *precipitated sulfur* (2 to 5 per cent) in cold cream is a useful combination An *astringent lotion* of considerable value is

| | |
|-----------------------|----------------|
| ℞ Precipitated Sulfur | 12 0 |
| Powdered Camphor | 0 5 |
| Powdered Tragacanth | 1 5 |
| Lime Water | |
| Water | aa qs ad 120 0 |

Care must be taken not to irritate the skin because the instigation of a dermatitis seems particularly easy in the patient with a seborrheal skin

Roentgen therapy in experienced hands often produces lasting benefit but dosage must be carefully regulated *Ultraviolet light* is of benefit in many cases

The *oily scalp* must be treated at the same time Shampoos with soap and warm water can be performed once or twice weekly and more often in especially severe cases Ordinary soap may be used or *Tincture of Green Soap* Tar soaps or Sulfonated Oil preparations *Ointments* or *lotions* may be prescribed containing as active principles *sulfur resorcin mercury* or *salicylic acid* Ointments are rubbed in with the fingers while lotions may be sprinkled on the scalp and then rubbed in with the fingers or a soft toothbrush Representative formulas follow

| | |
|---|---------------|
| ℞ Salicylic Acid | 1 5 |
| Precipitated Sulfur | 3 0 |
| Benzonated Lard | qs ad 60 0 |
| ℞ Resorcinol | 0 5 |
| Precipitated Sulfur | 1 5 |
| Anhydrous Lanolin | |
| Petrolatum | aa qs ad 60 0 |
| ℞ Ointment of Ammoniated Mercury (3 to 10 per cent) | |
| ℞ Resorcinol Monacetate | 8 0 |
| Bichloride of Mercury | 0 0 |
| Triethanolamine | 10 0 |
| Spirits of Lavender | 80 0 |
| Alcohol (70 per cent) | qs ad 240 0 |

(To this may be added 10 0 cc of castor oil or sweet almond oil if the scalp is dry)

Removal of foci of infection should be practiced. It is said that patients are greatly benefited by residence in a warm dry climate. Local treatment of the skin is valuable especially gentle massage, baking contrast baths, diathermy and active and passive exercises should also be performed.

SEBORRHEA

Seborrhea is a noninflammatory condition of unknown causation which manifests itself as an oily (seborrhea oleosa) and dry (seborrhea sicca) variety. Either of these may become inflammatory and lead to *seborrheic dermatitis* (seborrheic eczema). The areas most commonly affected by these conditions are the *scalp* and *face* and the *interscapular* and *presternal regions*. In these portions of the body are located the greatest concentrations of sebaceous glands.

Etiology—The cause of seborrhea is not known. It occurs in both sexes, brunettes and Negroes in whom the condition is most often found are particularly affected by the oily variety while blonds show the dry variety. The affection may appear in infancy but is most common during the pubertal period when the sebaceous glands become most active. Prolonged illness, sedentary occupation, poor scalp hygiene and the wearing of tight poorly ventilated hats are said to be predisposing factors. It is most probable that they play only a small part if any in the disease.

There are a number of suggested etiologic agents which may have more than a theoretical relationship. The tendency for the condition to appear at puberty like *acne vulgaris* poses the possibility of *endocrine origin*. In acromegaly there frequently is noted an oily seborrhea. The possibility of an underlying disturbance of the *metabolism of fat* has been broached although there is little evidence to encourage this idea. *Heredity* may be a factor since there is an undoubted tendency for this condition to prevail in certain families. Recent investigations both in animals and on humans raise the question of a *vitamin deficiency* as the causal mechanism. A seborrhea like condition occurs in rats whose diet is deficient in certain components of the B complex, namely *biotin*, *pyridoxine*, *pantothenic acid* and *riboflavin*. These substances are naturally richest in liver and yeast.

The belief that a disturbance of the *central nervous system* may be concerned is suggested by the syndrome of *postencephalitic seborrhea*. Following encephalitis lethargica many patients develop a very marked oily secretion of the face and a dry scaling condition of the scalp. These individuals almost always show muscular rigidity, masklike facies and profuse salivation. Similar changes are sometimes noted in paralysis agitans and in patients with other cerebral lesions.

Clinical Manifestations—*Oily seborrhea* (seborrhea oleosa) manifests itself as an excessively greasy condition of the *seborrheal areas* (scalp, face, presteral and interscapular areas). The openings of the *follicles* may be *patulous* or may contain plugs of *sebum* which are easily expressed. Little or no redness and scaling are present. The oily film on the face readily entraps dust, soot and chemicals and may be a factor in the development of occupational dermatitis.

Dry seborrhea (seborrhea sicca) appears on the scalp as fine slightly greasy whitish scales and is commonly known as dandruff. It may be limited to the scalp or involve additionally the brows, neck and ears. The eyebrows may also show scaling. Scaling slightly pinkish circumscribed patches may occur on the arms and trunk. On the scalp the eruption may be widespread or patchy.

A crusted form of dry seborrhea is sometimes seen. On the face it appears as a diffuse pale yellowish waxy film spreading over the nose and adjacent cheeks and in the nasolabial folds. On the scalp it is

of large waxy yellowish scales which may mat together with the hair and form a thick dry crust Alopecia is frequently present

Treatment—The treatment of *oily seborrhea* must be directed at the scalp as well as the nonhairy surfaces The general health should be improved Fresh air sunshine regular exercise and a simple low fat diet are advised The addition of *liver yeast* and *vitamin B complex* to the diet is desirable Frequent washing of the skin with soap and water and for women a face powder containing sulfur are recommended Ordinary white soap may be used but in stubborn cases *Tincture of Green Soap* may be more satisfactory *Resorcin* and *sulfur* are effective remedies and may be prescribed as the *lotions* or *salves* which are used in *acne vulgaris* (p 3362) They should be used in low concentration at first as 1 per cent resorcin and 2 per cent sulfur and the strength increased if no irritation is produced *Salicylic acid* (1 to 2 per cent) and *precipitated sulfur* (2 to 5 per cent) in cold cream is a useful combination An *astringent lotion* of considerable value is

| | |
|-----------------------|-----------------|
| R Precipitated Sulfur | 12.0 |
| Powdered Camphor | 0.5 |
| Powdered Tragacanth | 1.5 |
| Lime Water | |
| Water | aa q.s ad 120.0 |

Care must be taken not to irritate the skin because the instigation of a dermatitis seems particularly easy in the patient with a seborrheal skin

Roentgen therapy in experienced hands often produces lasting benefit but dosage must be carefully regulated *Ultraviolet light* is of benefit in many cases

The *oily scalp* must be treated at the same time Shampoos with soap and warm water can be performed once or twice weekly and more often in especially severe cases Ordinary soap may be used or *Tincture of Green Soap* Tar soaps or Sulfonated Oil preparations *Ointments* or *lotions* may be prescribed containing as active principles *sulfur resorcin mercury* or *salicylic acid* Ointments are rubbed in with the fingers while lotions may be sprinkled on the scalp and then rubbed in with the fingers or a soft toothbrush Representative formulas follow

| | |
|---------------------|-------------|
| R Salicylic Acid | 1.5 |
| Precipitated Sulfur | 3.0 |
| Benzonated Lard | q.s ad 60.0 |

| | |
|---------------------|----------------|
| R Resorcinol | 0.5 |
| Precipitated Sulfur | 1.5 |
| Anhydrous Lanolin | |
| Petrolatum | aa q.s ad 60.0 |

R Ointment of Ammoniated Mercury (3 to 10 per cent)

| | |
|-------------------------|--------------|
| R Resorcinol Monacetate | 8.0 |
| Bichloride of Mercury | 0.2 |
| Triethanol mine | 10.0 |
| Spirits of Lavender | 80.0 |
| Alcohol (0 per cent) | q.s ad 240.0 |

(To this may be added 10.0 cc of castor oil or sweet almond oil if the scalp is dry)

| | |
|-----------------------|--------------|
| ℞ Resorcinol | 120 |
| Alcohol (70 per cent) | 800 |
| Rose Water | q.s. ad 2400 |

(For dark haired individuals)

These preparations should be applied to the scalp each night until the condition subsides and then less frequently. Since the disease tends to recur the prophylactic use of the medicament once weekly is not amiss.

The scalp can also be treated with *roentgen rays* although special caution in regards to dosage is necessary because of the possibility of causing *epilation*. *Ultraviolet irradiation* is of value in some cases and may be used two or three times weekly.

Dry seborrhea may be treated in a similar manner to the above, except that ointments are generally preferable to lotions and shampooing should be done less frequently.

SEBORRHEIC DERMATITIS (SEBORRHEIC ECZEMA)

Seborrheic dermatitis is the inflammatory sequel of seborrhea (*oleosa* or *sicca*). Basically the latter conditions are functional disorders of the sebaceous glands unattended by inflammation. When clinical evidences of inflammation supervene the diagnosis of seborrheic dermatitis is made.

Etiology—The agents which are responsible for the conversion of seborrhea into seborrheic dermatitis are not exactly clear. *Microbes* of various sorts are blamed. These include pyogenic cocci, bacilli and especially the *Pityrosporum ovale* (bottle bacillus). The *pyrosporum* is a yeast like fungus which is very frequently present in this condition but opinion as to its causal relationship differs. By some it is considered the actual cause by others a harmless and innocent secondary invader. Some observers find a more than causal relationship between seborrhea and psoriasis and regard the former as merely one phase of the latter.

Heat, friction and moisture in a seborrheal region are definite predisposing factors. The composition of *fatty acids* and other materials may be contributory. The disease is most prevalent and at its worst in the winter. Seborrheal skin is notoriously susceptible to irritation and inflammation may be induced by external applications.

Localization—Seborrheic dermatitis is most common in the so called *seborrheal areas* of the scalp, face, presternal and interscapular regions. It has a marked tendency to spread and involve the ears, the eyelids, the axillae, the inguinal region and the umbilicus. It may affect the trunk, the extremities and in rare instances may become generalized. The scalp is almost always the first area affected. It may remain localized here or it may spread by contiguity to the forehead, neck and ears. In time remote involvement may arise in the common seborrheal sites. Spread may be slow and insidious over a period of many years or it may be almost explosive in its suddenness and rapidity.

More or less *alopecia* may be present with seborrheic dermatitis of the scalp. A persistent *blepharitis* is a not uncommon concomitant.

Clinical Varieties—The common clinical varieties of the disease are the *dry reddened*, scaly, macular or papular form and the *moist* eczematoid type which appears in the sites of predilection for intertrigo (the post auricular region, the umbilicus, the axillae and the groins).

In the *dry scaly form* there are yellowish scales, greasy and adherent or dry and flaky arising from rounded or irregular pinkish patches. The areas extend centrifugally and may fuse and form large plaques which are

rounded irregular or shaped like a flower petal (petaloid) The border of the lesion is the active zone and may be macular or slightly papular and reddish in color The central portion is yellowish and scaly On the scalp lesions may be discrete or they may enlarge so that the entire region is covered with the eruption On the body the lesions are more often discrete few in number and easily identified In the nasolabial fold it appears as a diffuse reddened area with the characteristic scale There may be scaling of the auricle and the proximal portion of the external auditory canal Isolated dry reddened scaly coin sized patches may be present on the arms Scaling may be present in the eyebrows and beard

The *moist intertriginous form* of seborrheic dermatitis is less common. It occurs most frequently behind the auricles and in the axillary umbilical and inguinal regions It begins as scaly red areas which are changed to moist edematous oozing surfaces by the action of moisture friction and heat The lesion resembles closely the picture produced by *dermatitis intertrigo* and *dermatitis venenata* in these locations Because of its marked irritability and the associated burning pain and itching any movement of the part may be extremely painful

Diagnosis—Seborrheic dermatitis must be differentiated from dermatitis venenata atopic dermatitis psoriasis pityriasis rosea dermatitis intertrigo and fungus diseases *Dermatitis venenata* is more inflammatory more acute frequently distinctly vesicular and more pruritic Especially in the moist variety occurring in the axillae doubt may arise as to differentiation In this location dermatitis venenata (caused by dress shields anhydrotics chemical depilatories and dress dyes) is not so likely to spread over the entire axilla as is the seborrheic condition the apex of the axilla usually being free of eruption However in every case dermatitis venenata as the sole cause or a supplementary factor should be considered and eliminated

Atopic dermatitis usually attacks somewhat different regions is dry lacks the greasy scale and the history of atopy is suggestive Pityriasis rosea appears on the trunk but the scalp is clear It has an acute onset and a self limited course The lesions are ovate with their long axis in the skin cleavage lines and they lack the greasy scale *Psoriasis* confined to the scalp offers great difficulty but the lesions are more infiltrated usually more discrete and the scale is dry and silvery On the body psoriasis attacks the extensor surfaces of the extremities the scales are never greasy and confirmatory evidences may be present in the finger nails In *dermatitis intertrigo* the absence of seborrheic lesions on the scalp and in other regions of the trunk is helpful *Tinea corporis* has a vesicular margin a clear center and the fungi are demonstrable *Momiasis* of the folds usually shows isolated satellite pustules off to one side of the main lesion It need not be bilaterally symmetrical and the yeast organisms can be found

Treatment—Systemic therapy is not of great value in seborrheic dermatitis but those measures recommended for seborrhea (p 3431) may be applied Injections of *liver extract* or *vitamin B complex* in the refractory cases of seborrheic dermatitis are highly regarded by some physicians

The treatment of the local condition is determined by the clinical character of the eruption In general in the dry scaly varieties it may be

vigorous, while in the moist inflamed varieties it must be bland simple and soothing. The scalp is looked upon as the fountainhead of the disease and must be treated in every case (p. 3444).

The *dry scaly variety* responds fairly readily to comparatively simple therapy. The following formulas may be used:

| | | |
|---|---------------------|-----------------|
| R | Salicylic Acid | 1.0 |
| | Precipitated Sulfur | 3.0 |
| | Cold Cream | q.s. ad 60.0 |
| R | Resorcinol | 1.0 |
| | Precipitated Sulfur | 3.0 |
| | Lanolin | |
| | Petrolatum | aa q.s. ad 60.0 |
| R | Salicylic Acid | 1.5 |
| | Ammoniated Mercury | 3.0 |
| | Petrolatum | q.s. ad 60.0 |
| R | Salicylic Acid | 1.5 |
| | Oil of Cade | 3.0 |
| | Precipitated Sulfur | 3.0 |
| | Petrolatum | q.s. ad 60.0 |

(For more infiltrated and resistant lesions.)

In the more chronic forms when response to topical treatment is slow or absent the use of *ultraviolet light irradiation* is often beneficial. *Roentgen therapy* is also useful in the refractory case. These modalities are of particular value in the occasional instance wherein the skin tolerates greasy substances poorly. Some of these show actual aggravation of the skin lesions after the use of simple topical medicaments.

The *moist inflammatory type* of seborrheic dermatitis which attacks the flexural regions must be treated gently. When the skin is reddened and oozing and movement is painful *bed rest* is advisable. *Wet dressings* are then the treatment of choice and boric acid solution or diluted Burow's solution (1:20) may be used. At night and during the daytime between applications of the wet dressings *calamine liniment or lotion* is applied. When the acute symptoms have subsided a *bland ointment* may be used such as ointment of *zinc oxide* or equal parts of *boric acid ointment* and *cold cream*. Later such active remedies as *precipitated sulfur* 1 to 5 per cent *Ammoniated Mercury* 1 to 3 per cent or *Liquor Carbonis Detergens* 3 to 5 per cent may be added. The sulfur and mercury should never be combined. *Roentgen therapy* is often extremely helpful in this variety of seborrheic dermatitis.

The blepharitis is treated with the ointment of 1 per cent *yellow oxide of mercury*.

CHAPTER 151

DISTURBANCES OF THE HAIR

Abnormalities of Hair Distribution

Feminization in the Male

Masculinization in the Female

Localized Hypertrichosis

Vinism

Alopecia

Congenital Alopecia

Monilethrix

Symptomatic Alopecia

Alopecia from Local Dermatoses

Alopecia from Roentgen Ray

Alopecia from Mechanical Trauma

Alopecia Cicatricata

Folliculitis Decalvans

Postfebrile Alopecia

Syphilitic Alopecia

Alopecia in Endocrinopathies

Drug Alopecia

Senile Alopecia

Premature Alopecia

Alopecia Areata

Canities

Atrophies of the Hair

Monilethrix

Trichorrhexis Nodosa

Leptotrich

The downy lanugo present at birth is soon cast off and the scalp hair eyelashes and eyebrows remain as the only hirsute adornments until the age of puberty. At this time the familiar male and female *secondary sex characteristics* become prominent and these determine the appearance of the individual for the greater part of adult life.

The *growth of hair* is constant as new hairs grow from the papillae when the old are shed. Each scalp hair increases daily $\frac{1}{2}$ mm. in length and its average life approximates four years. Blondes have the greatest numerical growth of scalp hair brunettes next and redheads the least.

Cosmetic Aspects—The hairs of the head and face are important determinants of feminine and masculine appearance. They do much to enhance or mar the beauty of *women* who lavish much effort and money upon their hirsute charms and defects. Those who have curly hair employ hair straighteners while the straight haired lasses expend their time and money on permanent waves, curlers and bangs of various sorts. Blondes bleach their hair to a more aureate hue, brunettes add henna to give their hair the glad appearance while the darker haired effect a raven tress. Women with gray hair resort to dyeing when they would appear young and blueing when they seek to accentuate the whiteness. Commercial hair institutes, beauty parlors and the cosmeticians exploit these vanities and

at exorbitant prices sell bleaches dyes blues straighteners curlers and brilliantine all of which on final analysis are simple of composition and easily and inexpensively reproduced See p 3138

The male contingent is no less hair conscious The daily shave is a necessary chore since beard and moustache went out of style and because it is no longer fashionable to hide a weak chin acne scars or a thin lip with a formidable beaver males are compelled to rely for their dominance upon character traits rather than the patriarchal hirsute adornment

The vanity of the male is best exhibited by his reaction to baldness Men are amused by the gullibility of the female relative to scalp treatments but will go to great extremes in their own efforts to avoid and cure baldness

The Practitioner and Problems of the Hair—The interested practitioner may do much to improve the public attitude toward the scalp and hair Much more can be accomplished by simple *shampoos* (p 3141) and *hair lotions* (p 3141) than by all the ministrations of the commercial hair experts Transitory conditions such as certain types of alopecia (p 3140) require only that the afflicted impose no further damage Permanent alopecias should be accurately prognosticated to save the time and money so wastefully expended by those whose vanity is equalled only by their credulity

Classification of Hair Disorders—The principal abnormalities of hair include disorders of hair distribution alopecia canities and atrophy

ABNORMALITIES OF HAIR DISTRIBUTION

Abnormalities of hair distribution have diagnostic and cosmetic significance Feminization occurs in males masculinization with hypertrichosis and hirsuties in the female virilism is seen in either sex and localized hypertrichoses are observed in various localities

FEMINIZATION IN THE MALE

At puberty with the development of secondary sex characteristics the male acquires a dark hair growth in the mustache and beard areas in the axilla on the sternum and in the pubic region In the latter site the conformation is that of a triangle with its apex at the umbilicus and base at the pubis

Some boys not strikingly masculine in appearance lag in their hirsute development within physiological limits They tend to have a sparse hair growth, shave infrequently and never completely develop the male escutcheon in the pubic region These boys are not necessarily abnormal nor do they lack virility or the heterosexual attitude

Endocrinologic Implications—Endocrinologists stress these variations and intimate that the feminized male suffers from hypoplasia of anterior pituitary adrenal cortical or gonadal functions However these generalities are not to be taken too literally since the body economy of these young men may not suffer and attempts at alterations of the endocrine balance are fraught with great danger of harm and small prospect of significant achievement

Sexuality—Psychotherapeutically it is important to reassure these boys that they are not sissies They should be prevented from acquiring

abnormal companionships and habits or from developing a compensatory masculine aggressiveness. Occasionally males who have a pathologically feminine appearance reveal a dislocation of the blood *estrogen-androgen* ratio toward the feminine side. These boys are in great danger of succumbing to homosexuality.

Treatment—At the present time endocrine therapy is of little avail. Injections of *androgen* are as likely to cause atrophy of the testes as stimulation. More important is frank conversation with the boy or his father, deliberate attempts to encourage feminine contacts and a firm policy of avoiding strictly masculine schools and occupations known to be liberally infiltrated with homosexuality (p 2412).

DIFFERENTIAL DIAGNOSIS OF

Dermatoses of the Beard

See also Dermatoses of the Face p 3286

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|------------------------------|---|
| <i>Alopecia areata</i> | p 3445 |
| <i>Pustular folliculitis</i> | A superficial staphylococcal infection of hair follicles. Identify pathogen (p 3249). |
| <i>Seborrheal dermatitis</i> | Reddened and scaly macular areas or moist greasy plaques. Associated with seborrhea of the scalp (p 3432). |
| <i>Sycosis vulgaris</i> | Chronic deep staphylococcal inflammation of hair follicles (p 3249). |
| <i>Tinea barbae</i> | Fungous infection of hair follicles. Note tender red infiltrated swellings. Identify pathogen (p 3304). |
| <i>Trichorrhexis nodosa</i> | Dystrophy of the hairs of the beard. Note nodular swellings of the shafts of the individual hairs (p 3449). |

MASCULINIZATION IN THE FEMALE

The female hair pattern conventionally exhibits absence of black bristle like hairs in the moustache and sternal regions, although axillary hairs do develop. The female escutcheon in the pubic region is that of a horizontal line without extension toward the umbilicus.

Hypertrichosis and Hirsuties—Variations in hair development occur within physiological limits. Masculinization of the female is noted frequently in younger girls and women who may have a dark moustache, a few black bristles on the chin and in the region of the breasts, some presternal growth and a triangle extending toward the umbilicus in the pubic region. Such women have a tendency to develop acne vulgaris (p 3358) and this together with the superfluous hairs causes some dismay to the afflicted and fills the coffers of the cosmeticians. The masculinized females vary in the degree to which they exhibit their abnormalities and in their reaction. More often than not these young women are exceedingly sensitive about their appearance and require tactful and skilful management from a dermatological and psychological standpoint.

When the masculinization occurs as it so frequently does at the menopause or after the problem is not difficult but the appearance of *hypertrichosis* in young women between sixteen and twenty one when they desire to be most alluring and attractive is a formidable challenge. These young women are otherwise completely normal. Often there is a family history of a similar hair growth. Undoubtedly, the abnormality is due to a *dislocation in the ratio between circulating male and female sex hormones in favor of the former*. Perhaps this explains the greater tendency of these young women to develop *acne vulgaris* (p 3358).

Treatment—The management of *hypertrichosis* in the female involves local treatment, endocrine therapy and psychotherapy.

Local Therapy—The local therapy of *hypertrichosis* may be managed by many techniques. Larger hairs are removed mechanically with *tweezers*. They can be rubbed down with pumice stone after they have been shaven or cut off. In point of fact the prolonged use of the pumice stone may bring about permanent atrophy of the hair follicles. Downy hairs such as profusely appear on the moustache can be *bleached* with peroxide (p 3110). *Epilation with wax* can be successfully practiced with all but stiffest bristles. The procedure is moderately painful but few things exceed the stoicism of vanity.

Most fastidious women perform a *dry shave* for arms, legs and axillae. This is perhaps the safest and most satisfactory method since the use of *chemical depilatories* (p 3139) is apt to lead to infection, contact dermatitis and axillary gland abscess.

Exposure to *roentgen ray* for permanent epilation is dangerous and shunned by reputable dermatologists. The amount of therapy required is almost certain to lead to *radiodermatitis* (p 3179) with all of its dread sequelae to the patient and medicolegal jeopardy to the operator.

When simple remedies cannot cope with the situation the patient may be referred to the expert for epilation by *electrolysis* (p 3792). He may employ the galvanic current or a weak electrocoagulating current. These procedures are tedious and moderately painful. In experienced hands they lead to permanent epilation without visible scarring. In expert use of electrolysis results disastrously for patient and operator alike since permanent scarring disfigures the former and threatens the operator with legal consequences.

Endocrine Treatment—The conscientious practitioner discourages the injection of potent endocrine products for cosmetic purposes. He prevents fastidious and desperate patients from receiving unwarranted and perhaps harmful substitution therapy from overzealous overenthusiastic endocrinologists since the injection of a hormone product of sufficient potency to alter hair growth is capable of profound and often untoward effects in other organs of the body.

Psychotherapy—Psychotherapy is used as an adjunct to local treatment. It is futile to attempt to understate the extent and degree of the hirsute blemish. The practitioner should accept the patient's evaluation of the extent of the cosmetic trauma and approach the problem with the same care and concern as he would a major therapeutic problem.

Inexpertly handled by the method of belittling or scolding these young women react by becoming surly, inaccessible and solitary or, at the other

DIFFERENTIAL DIAGNOSIS OF

Alopecia (Baldness)

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|------------------------|--|
| Acrodermia | In infants. With redness and swelling of hands and feet. Try vitamin B complex (p 3145) |
| Alopecia areata | Psychogenic. May be urticarial. Look for excoriation on posterior hairs. May disappear and reappear spontaneously (p 3445) |
| Alopecia cicatricata | Irregular patches. Scalp area atrophic. Try vitamin A (p 3412) |
| Drugs | Especially thallium used locally or as urticaria (p 760 3143) |
| Exfoliative dermatitis | With scaling of entire skin. Often pharmacodynamic following antler therapy (p 3383) |
| Favus | Look for cup-shaped yellow crusting. Isolate fungus (p 3304) |
| Folliculitis decalvans | Circumcribed areas of pustulation. Followed by scarring and permanent baldness (p 3421) |
| Ketion celsi | Fungous infection resulting in permanent baldness (p 3303) |
| Lepidoma | A trichomycosis of axilla. Note brown nodules on hair (p 3453) |
| Lupus erythematosus | With spread of facial dermatitis. Note red shiny skin and keratotic prolongations (p 3355) |
| Lupus vulgaris | With spread of facial dermatitis. Note apple jelly nodules with ulceration. Get biopsy (p 3263) |
| Monilethrix | Hereditary partial alopecia. Hair dry and short. Constrictions seen with hand lens (p 3440) |
| Postfebrile | Especially after pneumonia and typhoid fevers. Observe spontaneous regrowth (p 3443) |
| Premature alopecia | See p 3444 |
| Radodermitis | Following x-ray and radium therapy (p 3179) |
| Scleroderma | In association with generalized dermatosis producing hide-bound skin (p 3427) |
| Seborrhea | With dry dandruff or greasy variety (p 3430) |
| Senile alopecia | See p 3144 |
| Syphilis | Rarely observed. May be areticular in treated cases (p 340) |
| Tinea capitis | Trichomycosis. Note broken-off hairs and local inflammatory reaction (p 3294). Isolate fungus. |
| Trichorrhexis nodosa | Note brown nodules on shaft of hair due to dystrophy (p 3443) |
| Trichotillomania | Psychogenic disorder in which hairs are plucked out (p 3234) |

extreme they are impelled to emphasize their masculinity and become threatened by engulfment in homosexuality (p 2412)

When the masculinization occurs as it so frequently does at the menopause or after the problem is not difficult but the appearance of *hypertrichosis* in young women between sixteen and twenty one when they desire to be most alluring and attractive is a formidable challenge. These young women are otherwise completely normal. Often there is a family history of a similar hair growth. Undoubtedly, the abnormality is due to a dislocation in the ratio between circulating male and female sex hormones in favor of the former. Perhaps this explains the greater tendency of these young women to develop *acne vulgaris* (p 3358).

Treatment—The management of hypertrichosis in the female involves local treatment, endocrine therapy and psychotherapy.

Local Therapy—The local therapy of hypertrichosis may be managed by many techniques. Larger hairs are removed mechanically with *tweezers*. They can be rubbed down with pumice stone after they have been shaven or cut off. In point of fact the prolonged use of the pumice stone may bring about permanent atrophy of the hair follicles. Downy hairs such as profusely appear on the moustache can be *bleached* with peroxide (p 3119). *Epilation with wax* can be successfully practiced with all but stiffest bristles. The procedure is moderately painful but few things exceed the stoicism of vanity.

Most fastidious women perform a *dry shave* for arms, legs and axillae. This is perhaps the safest and most satisfactory method since the use of *chemical depilatories* (p 3139) is apt to lead to infection, contact dermatitis and axillary gland abscess.

Exposure to *roentgen ray* for permanent epilation is dangerous and shunned by reputable dermatologists. The amount of therapy required is almost certain to lead to *radiodermatitis* (p 3179) with all of its dread sequelae to the patient and medicolegal jeopardy to the operator.

When simple remedies cannot cope with the situation the patient may be referred to the expert for epilation by *electrolysis* (p 3792). He may employ the galvanic current or a weak electrocoagulating current. These procedures are tedious and moderately painful. In experienced hands they lead to permanent epilation without visible scarring. In expert use of electrolysis results disastrously for patient and operator alike since permanent scarring disfigures the former and threatens the operator with legal consequences.

Endocrine Treatment—The conscientious practitioner discourages the injection of potent endocrine products for cosmetic purposes. He prevents fastidious and desperate patients from receiving unwarranted and perhaps harmful substitution therapy from overzealous overenthusiastic endocrinologists since the injection of a hormone product of sufficient potency to alter hair growth is capable of profound and often untoward effects in other organs of the body.

Psychotherapy—Psychotherapy is used as an adjuvant to local treatment. It is futile to attempt to understate the extent and degree of the hirsute blemish. The practitioner should accept the patient's evaluation of the extent of the cosmetic trauma and approach the problem with the same care and concern as he would a major therapeutic problem.

Inexpertly handled by the method of belittling or scolding these young women react by becoming surly, inaccessible and solitary or at the other

SYMPTOMATIC ALOPECIA

Symptomatic alopecia may result from local and systemic cause. Among the *local conditions* producing baldness are the dermatoses, exposure to roentgen ray, mechanical trauma, and the rare alopecia cicatrizzata and folliculitis decalvans. The *systemic causes* for symptomatic alopecia may be related to generalized infectious or debilitating disease, endocrine dysfunctions, or the ingestion of certain drugs.

Alopecia from Local Dermatoses—The dermatoses produce local destruction of the hair follicles resulting in a *circumscribed alopecia* in the areas where the affected portion of the scalp is scarred and atrophic. Important causes for the production of these scars include trauma, burns, the applica-

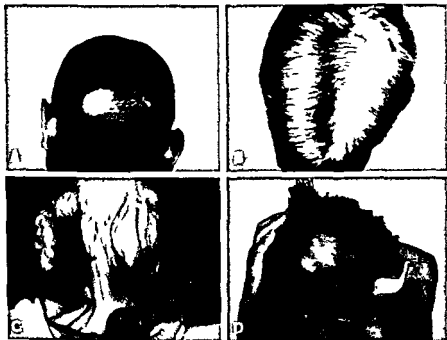


Fig 1007—A Alopecia areata B Alopecia areata (en coup de sabre) C Alopecia areata D Alopecia syphilitica

tion of chemicals, pyogenic infection, scleroderma, lupus vulgaris, lupus erythematosus, tertiary syphilis, and favus. *Exfoliative dermatitis* (p 3383) is often accompanied by partial or complete alopecia of the scalp and shedding of the nail. Partial alopecia in children is usually due to *fungous infections* of the scalp and hair (p 3294). Permanentarring rarely results except in those instances of *kerion celsi* where a fungus of animal origin produces the inflammatory condition and in *favus* (p 3304).

The *prognosis* in baldness resulting from dermatoses is unfavorable except in exfoliative dermatitis.

Alopecia from Roentgen Ray—Exposure of a hairy region to the *roentgen ray* or *radium* will cause *epilation* if the necessary dosage is given. Temporary epilation by roentgen rays is used as the treatment of choice

LOCALIZED HYPERTRICHOSIS

Localized hypertrichosis is generally congenital. It occurs as part of a *hairy pigmented nevus* (p 2204). It is also observed over the site of a *spina bifida* (p 2820).

VIRILISM

The sudden appearance of virilism in male or female gives rise to the suspicion of serious organic disease. Often the disturbance is due to a neoplasm of endocrine origin. The culpable tumors include *carcinomas of the adrenal cortex* (p 1268), *arrhenoblastomas of the ovary* (p 2515), *basophilic adenomas of the pituitary* (p 1159) and *malignancies of the testes* (p 2442).

ALOPECIA

Alopecia may involve one or more circumscribed areas of the scalp, the whole scalp or the entire body. It may be permanent or temporary and results from a variety of internal and external causes of local or systemic origin. It is the practitioner's duty to establish the variety and cause of the alopecia and render an appropriate prognosis. Thus conservative treatment will suffice to correct a temporary baldness and expensive useless therapy must be avoided in the permanent types.

The varieties of baldness include congenital alopecia, symptomatic alopecia, senile alopecia, premature alopecia and alopecia areata.

CONGENITAL ALOPECIA

Congenital alopecia is a rare condition often accompanied by other ectodermal defects (p 3150) such as absence of the nails (anonychia) and faulty development of the teeth. Hereditary influences and especially those deriving from consanguinity of parents are factors in many instances. There is partial or complete failure of development of the hair follicles which may be universal or confined to circumscribed areas. The hair may be absent at birth or present in a modified form later falling out and never regrowing. The scalp is most commonly affected and may show a scant growth of imperfect hair later in life or remain completely and persistently bald.

Treatment is unsatisfactory although the application of stimulating lotions of *Resorcin* (p 3126) or *Mercury* (p 3121) is recommended. In some instances a growth of hair occurs spontaneously later in life.

MONILETHRIX

Monilethrix is a rare hereditary disease of the scalp hair in which partial alopecia is constantly present. It appears in childhood and may involve the whole scalp or only a part. The hairs are dry, dull and short. When examined with a lens they are seen to be constricted at regular intervals. The beaded hairs snap easily at the sites of constriction. *Keratosis pilaris* as seen in vitamin A deficiency (p 3236) almost always coexists over the occipital and nuchal regions. Partial alopecia due to atrophy of hairs is the rule.

There is no satisfactory therapy but large doses of vitamin A 50 000 to 200 000 units daily may be tried.

10 per cent *Sulfur Ointment* or 10 to 20 per cent *Sulfathiazole Ointment* Roentgen therapy is reserved for obstinate examples which fail to respond to simpler treatment Large doses of *vitamin A* (100 000 to 200 000 units daily) may be tried

POSTFEBRILE ALOPECIA

Postfebrile alopecia is characterized by a sudden and rapid shedding of the scalp hair during convalescence from a severe febrile disease It usually occurs six to ten weeks after the height of the fever Alopecia may follow any prolonged pyrexia (p 30) but it is most often observed after *typhoid fever* (p 225) *severe influenza* (p 396) *erysipelas* (p 162) and the *exanthematic diseases* Alopecia may also occur in the course of a chronic disease such as *anemia diabetes cachexia* and *tuberculosis*

Alopecia after fevers is a self limited condition and in practically every instance the hair regrows spontaneously This is true to a somewhat lesser extent of chronic debilitating diseases Regrowth of the hair occurs after the general health is restored although in older persons with premature alopecia the restitution may be incomplete and the progress of the preexisting alopecia may be hastened

Therapy consists of *shampoos* (p 3441) the application of *stimulating lotions or ointments* (p 3445) and exposure to *ultraviolet light* (p 3794)

SYPHILITIC ALOPECIA

Alopecia associated with the *secondary stage of syphilis* appears between the fourth and the sixth month after the infection is acquired and involves the scalp and eyebrows There is a partial patchy alopecia producing the well known moth-eaten appearance In this variety of syphilitic alopecia the hair returns whether antisyphilitic treatment is given or not (Fig 1007)

ALOPECIA IN ENDOCRINOPATHIES

Alopecia may be a consequence of *endocrine dysfunction* In *myxedema* (p 1193) there is scantiness of the scalp hair and partial or complete loss of the outer third of the eyebrows In *chronic tetany* (p 1232) due to hyperparathyroidism there may be partial alopecia of the scalp Patients with *adiposogenital dystrophy* (p 1166) reveal a sparsity of body hair with female distribution The *eunuchoid* individual and true *eunuch* show a relatively normal growth of hair on the scalp but a thinning or absence of hair in the beard and on the body

DRUG ALOPECIA

Drug alopecia is caused by the oral administration of *thallium acetate* which produces epilation in ringworm of the scalp The use of thallium as a depilatory in creams to be applied locally is obviously based upon the erroneous concept of its action The systemic use as a depilatory is to be condemned because of its potential toxicity and the lesser danger and greater safety of other measures (p 3792) The drug acts through the involuntary nervous system upon the endocrine glands and effects a defluvium of the hairs of the scalp and the outer third of the eyebrows Hair elsewhere on the body is unaffected The alopecia produced is temporary regrowth occurring usually in two to four months

for certain types of ringworm infection of the scalp hair. When roentgen rays or radium are applied in higher than epilating dosage the alopecia may be permanent. This eventuality must be anticipated and the patient informed when large dosages are required in the treatment of malignant neoplasms, lupus vulgaris or angiomas.

The prognosis in roentgen epilation varies with the dose. It is unaffected by treatment.

Alopecia from Mechanical Trauma—Temporary alopecia can occur from mechanical trauma to the hair. Prolonged rubbing of the hair induces localized incomplete alopecia as observed in the occipital region of young infants due to the constant movement of the head against the mattress or pillow. Prolonged scratching produces a similar effect. In neurotic persons afflicted with *trichotillomania* (p. 3234) the hair is torn out with the fingers. This hair loss is generally irregular and the scalp and hair are otherwise found to be perfectly normal. Recovery follows discontinuance of trauma.

Alopecia Cicatrisata—*Alopecia cicatrisata* (*pseudopelade*) is a rare condition in which rounded or irregular patches of permanent alopecia result from an idiopathic atrophic and scarring process. It is more common in males and practically always appears in adult life between the ages of twenty five and fifty. There are usually multiple areas of involvement but the vertex and occiput are the commonest sites of the affection. The spots are usually less than dime sized, whitish, seldom reddened and the skin is thinned, smooth, shiny, atrophic and free from follicular orifices. Often there are dilated follicles which contain horny plugs (*keratosis pilaris*) within the area and especially at the margin.

The outlook is indefinite in that the disease may spontaneously cease its progress. Unfortunately in other instances it proceeds until a considerable amount of permanent irrevocable alopecia results. There is never complete loss of scalp hair and the disease does not apparently occur in other areas.

Treatment is purely empirical and consists of improvement of the constitutional condition and the use of mild stimulating applications (p. 3445). The ingestion of vitamin A in large doses (100,000 to 200,000 units daily) may be of value especially in view of the associated *keratosis pilaris*.

Folliculitis Decalvans—*Folliculitis decalvans* is a rare affliction in which there are circumscribed areas of follicular pustulation followed by scarring and permanent alopecia. The cause of the condition is unknown but pyogenic organisms have generally been held responsible. A number of irregular groups of pustules, each pierced by a hair, may be present. When healing ensues the scalp becomes whitish, thinned, atrophied and scarred and the hair loss is permanent. Active pustular lesions may be seen about the scarred alopecic area.

The disease resembles *syccosis vulgaris* (p. 3249) which is seen chiefly in the bearded region. It is readily differentiable from *alopecia areata* (p. 3445). The presence of follicular pustules is the important determining point in eliminating other atrophic and scarring alopecias.

Treatment includes improvement of the general health and the local application of antiseptic substances such as Compound Quinolor Ointment (p. 3123), 10 per cent Ammoniated Mercury Ointment, Tincture of Iodine.

whereas the oily scalp is washed once or twice weekly Plain white soap castile soap tar soap or tincture of green soap is employed After the shampoo one of the ointments described below is massaged into the scalp while on other nights one of the pleasant lotions may be used There is no foundation for the belief that shaving the scalp stimulates hair growth

No 1 R

| | |
|---------------------|-----------|
| Salicylic Acid | 0.5-1.5 |
| Precipitated Sulfur | 10-30 |
| Cold Cream | qs ad 600 |

No 2 R

| | |
|---------------------------|--------------|
| Liquor Carbonis Detergens | 30-90 |
| Lanolin | |
| Petrolatum | aa qs ad 600 |

No 3 R

| | |
|---------------------|-----------|
| Resorcin | 0.5-3.0 |
| Precipitated Sulfur | 1.5-6.0 |
| Lanolin | 400 |
| White Petrolatum | qs ad 600 |

No 4 R

| | |
|--------------------------------|------------------|
| Ointment of Ammoniated Mercury | 3 to 10 per cent |
|--------------------------------|------------------|

These ointments are massaged into the scalp after the shampoo They may be applied at night once or twice weekly and removed the next morning with a cloth or brush or by shampoo On the intervening nights one of the following lotions is employed

No 5 R

| | |
|---|------------|
| Resorcinol Monoacetate | 40-80 |
| Bichloride of Mercury | 0.24 |
| Spirits of Lavender | 400 |
| Alcohol (70 per cent) | qs ad 2400 |
| (If the scalp is dry castor oil 80 cc is added) | |

No 6 R

| | |
|---------------------------|------------|
| Liquor Carbonis Detergens | 80 |
| Resorcinol Monoacetate | 40-80 |
| Alcohol (50 per cent) | qs ad 2400 |

No 7 R

| | |
|-------------------------|------------|
| Tincture of Cantharides | 100 |
| Tincture of Capsicum | 10 |
| Sp Vini Rect | 700 |
| Rose Water | qs ad 2400 |

No 8 R

| | |
|------------------------|------------|
| Resorcinol Monoacetate | 40-80 |
| Quinine | 20 |
| Castor Oil | 40 |
| Sp Vini Rect | qs ad 2400 |

ALOPECIA AREATA

Alopecia areata is a common form of hair loss which while usually confined to the head may affect any or all hairy regions In the rare examples of *alopecia universalis* not a strand of hair remains upon the body

Fatal accidental poisoning with thallium acetate has been reported from the ingestion of rat and ant poison

SENILE ALOPECIA

Senile alopecia occurs in most but by no means all men of advanced years. It is accompanied or preceded in most instances by graying of the hair (p 3448). The chief loss may be located upon the vertex of the scalp leaving a fringe of hair on the sides and may then proceed gradually backward from the frontal region. The bald area is smooth, shiny and taut and the hair follicles are atrophied. In women at or after the menopause there may appear some thinning of the scalp hair although true baldness is rare.

Senile alopecia is of unknown causation but it is likely that endocrine factors involved in the aging process play an important role. *Treatment* is rarely of value.

PREMATURE ALOPECIA

Premature alopecia (seborrheic alopecia) is the most common and distressing form of baldness. It is seen almost exclusively in the male although rare examples are encountered in females. The loss may commence on the vertex or in the frontal region or it may appear as recessions bilaterally in the temporal regions. It may progress rapidly until almost all of the hair is gone or it may proceed extremely slowly so that a considerable amount of hair is retained for many years.

Pathogenesis—The cause of premature alopecia is unknown. Familial trends are of great importance since premature alopecia of similar design is usually present in the male progenitors. Of the many accessory factors believed to be contributory, none seems to be consistently present. The allegedly culpable mechanisms include poorly ventilated or constricting head gear, prolonged exposure of the scalp to strong sunlight, excessive shampooing, infrequent shampooing, sedentary occupations and general ill health. The diametrically opposed concepts relative to the activating factors testify to the lack of knowledge about the entire subject.

The relationship of *seborrhea* (p 3430) of the dry scaling variety is much debated. Many regard dandruff as a most frequent cause of premature alopecia. Nevertheless, although alopecia and seborrhea often co-exist, a considerable number of baldheads reveal a surprisingly clean scalp and many patients with excessive dandruff never develop alopecia.

Prognosis—As opposed to the outlook in febrile alopecia and alopecia areata (p 3445), the prognosis for relief or arrest of presenile alopecia is unfavorable.

Treatment—The treatment of premature alopecia is almost exclusively local. Patients with an obvious endocrine dysfunction or constitutional defect require appropriate medication which in most instances has no effect on the baldness.

As local adjuvants, daily *massage* with the fingers or a firm brush, the wearing of light well ventilated hats, cessation of direct exposure to strong sunlight and avoidance of the hot air dryer in the beauty parlor are recommended. *Ultraviolet light irradiation* of the scalp may be beneficial. *Shampooing* should be performed according to the demands of the individual scalp. The dry scalp is washed once every two or three weeks.

whereas the oily scalp is washed once or twice weekly Plain white soap castile soap tar soap or tincture of green soap is employed After the shampoo one of the ointments described below is massaged into the scalp while on other nights one of the pleasanter lotions may be used There is no foundation for the belief that shaving the scalp stimulates hair growth

| | | |
|--------------------------------|------------------|-----|
| No 1 R | | |
| Sabcybe Acid | 0.5-1.5 | |
| Precipitated Sulfur | 10-30 | |
| Cold Cream | qs ad | 600 |
| No 2 R | | |
| Liquor Carbonis Detergens | 30-60 | |
| Lanolin | | |
| Petrolatum | aa qs ad | 600 |
| No 3 R | | |
| Resorcin | 0.5-3.0 | |
| Precipitated Sulfur | 1.5-6.0 | |
| Lanolin | 400 | |
| White Petrolatum | qs ad | 600 |
| No 4 R | | |
| Ointment of Ammoniat d Mercury | 3 to 10 per cent | |

These ointments are massaged into the scalp after the shampoo They may be applied at night once or twice weekly and removed the next morning with a cloth or brush or by shampoo On the intervening nights one of the following lotions is employed

| | | |
|--|-------|------|
| No 5 R | | |
| Resorcinol Monoacetate | 40-80 | |
| Bichloride of Mercury | 0.24 | |
| Spirits of Lavender | 400 | |
| Alcohol (70 per cent) | qs ad | 2400 |
| (If the scalp is dry ca 100 cc is added) | | |
| No 6 R | | |
| Liquor Carbonis Detergens | 80 | |
| Resorcinol Monoacetate | 40-80 | |
| Alcohol (50 per cent) | qs ad | 2400 |
| No 7 R | | |
| Tincture of Cantharides | 100 | |
| Tincture of Capsicum | 10 | |
| Sp Vini Rect | 00 | |
| Rose Water | qs ad | 2400 |
| No 8 R | | |
| Resorcinol Monoacetate | 40-80 | |
| Quinine | 2.0 | |
| Castor Oil | 40 | |
| Sp Vini Rect | qs ad | 2400 |

ALOPECIA AREATA

Alopecia areata is a common form of hair loss which while usually confined to the head may affect any or all hairy regions In the rare examples of *alopecia universalis* not a strand of hair remains upon the body

Clinical Appearance—Characteristically alopecia areata appears more or less suddenly as a solitary rounded coin sized *bald spot* upon the scalp. At times more than one spot may be observed at the outset. There are no premonitory symptoms or signs and the area is usually discovered accidentally by the patient, the barber or the hairdresser (Fig 1007).

The affected area is rounded and generally varies from the size of a dime to a fifty cent piece. Hairs may be entirely lost within the spot but often a few tiny tufts remain, these being usually loose and extractable with the gentlest traction. The hairs at the border of the lesion may be long and firm, a fact which is considered indicative of the cessation of active spread of the lesion; they may be broken off short and easily extracted, this being interpreted as a sign of continuing activity and spread. The short hairs have a thickened shaft which tapers down to a narrow pointed atrophic bulb, making them simulate an exclamation point. *Exclamation point hairs* are found at the border of an advancing lesion and within the denuded portion of a recently developed spot. The scalp itself is smooth, white and soft. It shows no evidences of inflammatory reaction nor is it atrophic.

Course and Prognosis—Alopecia areata is an unpredictable disease and the prognosis must always be guarded. Often one or several patches enlarge centrifugally to a certain maximum and then become stationary. Regrowth of the hair may occur within a year from the onset and be complete in three to six months. The hair that first appears is fine and blond but this is generally followed by a growth of thick hair, normal in every respect except that it is frequently white. The normal pigment develops later.

In the unfavorable case the original spots continue to enlarge and new ones appear. The spots coalesce and produce large patches of baldness. This process may continue until not a hair remains upon the scalp (*alopecia totalis*); there may ensue loss of hair in the eyebrows and eyelashes, the beard, axillae and pubic regions until all the body hair is shed (*alopecia universalis*). There is a marked tendency to recurrence of the condition; the hair returns only to be followed by new episodes of alopecia.

The prognosis bears a direct relationship to the age of the individual at the time the condition begins. The outlook is best in the young and becomes gradually worse as the age of the patient advances. This may be due to the natural senescence of the hair growing faculties rather than to any differences in the disease itself.

A certain number of patients with alopecia areata develop trophic changes in the nails (*leukonychia*, *fissuring*, *ridging*, *thimbling*). These signs indicate a poor prognosis.

Etiology—The exact cause of alopecia areata is unknown. It may appear at any age but is most common in children and young adults. It affects the sexes about equally. Among the etiologic agents which have been considered provocative of this condition are prenatal syphilis, hereditary parasitic infection, endocrinopathy and purely psychogenic mechanisms. The multiplicity of the alleged causative factors testifies to the fact that a clearcut case cannot be made against any one agency. Syphilis is readily eliminated by the clinical and serological findings. The hereditary factor is excluded by the history. As to a parasitic origin for the disease, neither clinical nor bacteriological data corroborate this suspicion. The

postulation of the existence of an *unknown virus* is in the realm of pure speculation

In an insignificant number of the afflicted a concomitant *abnormality of the endocrine organs* is discovered. However the overwhelming majority of patients reveal no such disorder. The assumption that some subclinical dysfunction not yet identified by our present methods is responsible constitutes a fanciful hypothesis which only adds confusion to the therapeutics.

The *psychogenic background* for alopecia areata has in our experience been impressive. The afflicted are tense high strung individuals in the midst of some superficial or profound emotional conflict. Certainly this hypothesis fits in with many of the clinical vagaries such as the youth of the patient, the tendency to recurrence, the absence of other demonstrable cause, the bizarre course of the disease, its tendency to recur, and the inconsistency and for the most part futility of therapeutic endeavors.

Diagnosis—The sudden appearance of alopecia areata, the rounded lesion with exclamation point hairs and the normal scalp are most distinctive features. *Ringworm of the scalp* (p. 3294) shows mild inflammatory reaction; the hairs are lustreless and broken off irregularly and laboratory studies reveal the fungi. *Syphilitic alopecia* (p. 3443) is practically always partial so that the areas are not completely bald but show the characteristic moth eaten appearance. Other manifestations of secondary syphilis are commonly present and the serologic tests of the blood are positive. Alopecia due to *destructive lesions* (lupus erythematosus, pyoderma, pseudopelade) is revealed by careful scrutiny of the scalp and the discovery of definite evidences of scarring and atrophy.

Treatment—It is extremely difficult to evaluate the efficacy of any form of therapy in alopecia areata. In the unfavorable case it is doubtful whether any form of therapy halts the progress of the disease. In favorable instances the regrowth of hair is most likely spontaneous.

The practitioner should neither promise that the hair will return nor warn that it will not return. He should declare simply that in most instances the hair of the scalp regrows in less than a year. The patient with total alopecia of the scalp, concomitant loss of eyebrows and eyelashes and generalized or universal alopecia is to be cautioned that several years may elapse before the hair returns and that there is a bare possibility that it will never return. *Alopecia of the beard* is notoriously refractory to any form of therapy.

The use of appropriate endocrine substitution therapy has been recommended for those patients afflicted with a definitive dysfunction. The administration of small doses of *thyroid extract* is suggested under any circumstance. Removal of *foci of infection* is urged as well as general measures to improve the health of the patient. The value of these procedures is doubtful however and no proof exists of their therapeutic effects on the condition. It is obviously futile and wasteful to exploit the patient by a needless tonsillectomy or tooth extraction which promises nothing. Any improvement that follows is more likely the result of spontaneous cure, suggestion or hope.

Psychotherapy is the most rational treatment procedure but results are as variable and inexplicable as with other modalities.

Local treatment should be employed despite skepticism as to its cura-

tive value If it does nothing else it occupies the patient's thoughts and buoys him with hope Stimulating and irritating lotions or ointments such as the following may be employed

R 1

| | |
|---------------------|--------------|
| Precipitated Sulfur | 30-60 |
| Betanaphthol | 20-40 |
| Lanolin | |
| Petrolatum | aa qs ad 600 |

R 2

| | |
|-------------|-----------|
| Oil of Cade | 30-60 |
| Cold Cream | qs ad 600 |

R 3

| | |
|------------------------|------------|
| Resorcinol Monoacetate | 40-80 |
| Bichloride of Mercury | 0.24 |
| Sp Odorat | 200 |
| Alcohol (70 per cent) | qs ad 2400 |

The *ointments* are massaged into the bald patches each night and cleansed away in the morning with mineral oil The *lotions* may be rubbed into the areas twice daily with a tooth brush or absorbent cotton These preparations are to be used only upon the scalp

A procedure of long standing used by many dermatologists is the weekly application by the physician of pure *carbolic acid* to the spots This is painted on the scalp which becomes blanched The excess is quickly removed with a cotton ball saturated with alcohol

Ultraviolet therapy has many adherents who believe it to be a most beneficial therapeutic modality The best results are said to be obtained when the exposure is pushed to the point of blister formation but the stage of simple erythema need not be passed

Alopecia of the beard is treated less stringently the skin being more sensitive Shaving should be persisted in if for no other reason than that the bald spots are made less noticeable Alcoholic lotions are preferred here and Formula No 3 above may be used

CANITIES (GRAYING OF THE HAIR)

The normal color of the hair is due to the presence of melanin Absence of pigment in the hair may be congenital or acquired The distribution may be circumscribed or diffuse The color of the hair becomes gray grayish white or snow white In senile canities the change is first seen in the scalp Later the hairs of the moustache and beard are involved and in advanced cases most of the body hair becomes affected In premature cases only the hair of the scalp shows the change

Ordinarily the graying of age begins between the thirty fifth and fortieth years while in the premature type it may commence before the twentieth year Senile graying in men begins earlier and is more intense than in women while the premature form is more common in women The hairs at the temples are generally affected first the spread then gradually involving the entire scalp

Etiology—The underlying cause of senile and premature canities is unknown The age of onset of the senile variety varies and may be related to the physiologic changes in the endocrine system It is not necessarily

accompanied by any deterioration of mental or physical vigor. There is a decided tendency for the premature variety to occur in certain families. Sudden graying of the hair has been reported from violent emotional experiences but authenticated examples are rare.

The most recent etiologic factor under investigation has been the *para aminobenzoic acid* factor of the *vitamin B complex* (p 622). While in experimental animals avitaminosis involving this substance results in pigimentary changes it does not follow that all cases of canities are of similar origin.

Any condition characterized by absent or deficient pigment formation in a hairy region may cause whitening of the hair. In *albinism* (p 1560) all the body hair is white. When a patch of *vittigo* (p 3404) occurs in the scalp, moustache, beard or elsewhere the hairs in this region become white and regain their normal pigmentation when the *vittigo* disappears. In *alopecia areata* the new hairs which grow into a bald spot are almost always white at first but eventually become dark.

Treatment—There is no recognized therapeutic procedure for the prevention or cure of graying of the hair. The administration of 100 to 200 mg of *para aminobenzoic acid* (p 627) given daily for two months has been claimed to restore the color in a majority of instances. Since these findings have not been fully confirmed and the toxicology of *para aminobenzoic acid* has been incompletely studied it may be best to defer this form of therapy for the present. Some investigators claim that *pantothenic acid* (p 626) which is concerned in the *achromotrichia* of rats is probably the agent needed for the treatment of human graying of the hair but this hypothesis too is unproved at the present time.

Until the subject is clarified the vitamins may be given in average doses. The patient who is unhappy with hoary locks may be advised to have recourse to hair dyes (p 3141).

ATROPHIES OF THE HAIR

A number of unusual conditions are seen in which the hair becomes atrophied as a result of changes in the shaft. Most of these are poorly understood but some have importance since they probably are brought about by the process of hairdressing.

Monilethrix—*Monilethrix* has been discussed (p 3340).

Trichorrhexis Nodosa—*Trichorrhexis nodosa* may appear in the male moustache and beard or in the hairs of the scalp of both sexes. When the hair is examined with a lens it shows nodular swellings along its shaft. The hair at these points becomes frayed out and brushlike fractures and breaks off. The scalp and hair root are entirely normal. The shortening of the hairs gives the scalp a baldish appearance although there is no actual loss. This condition is frequently accompanied by *trichoptilosis* in which the distal end of the hair reveals multiple longitudinal splitting.

While the cause of these conditions is probably not the same in every case it is believed that many are due to harsh treatment of the hair. The agencies which may be responsible are rough and excessively frequent brushing and rubbing, frequent hair waving, the use of hot air drying and the application of strong soaps and hair tonics.

The results of treatment are not particularly successful. It is advised

that the causative procedures be discontinued and the hair treated with olive or castor oil

Leptothrix—*Leptothrix* (*trichomycosis nodosa*) (p 3305) is a not uncommon condition affecting the hairs of the axilla. The hairs show pin head sized or slightly larger nodular masses of a brownish or yellowish color. At times the hairs are completely covered with this mass of fungous material. The skin is never affected. The hairs become brittle and dry and break off easily. The condition is practically always accompanied by hyperhidrosis.

Treatment consists of thorough soap and water washing of the affected areas and the application twice daily of an aqueous solution of bichloride of mercury (1:2000). Shaving the hair is the most beneficial procedure.

CHAPTER 152

DISEASES OF THE NAILS

Congenital Abnormalities

Anonychia
Mecroonychia
Macronychia
Pachyonychia

Trauma

Hangnail
Ingrown Nail
Subungual Hematoma

Neoplasms

Glomus Tumors
Melanocarcinomas
Epitheliomas
Fibromas
Angiomas

Pigmentation

Infections

Paronychia
Cranuloma Pyogenicum
Tuberculosis
Syphilis
Anaerobiasis

Verruca

Onychomycosis

Moniasis

Nail Changes in Cutaneous Diseases

Psoriasis
Contact Dermatitis
Exfoliative Dermatitis
Epidermolysis Bullosa
Alopecia Areata
Radiodermatitis

Dystrophies

Onychauxis and Onychogryphosis
Onychomadesis
Onycholysis
Onychorrhexis
Transverse Lines
Onychotrophia and Onychophaga
Fragilitas Unguium
Koilonychia
Leukonychia
Clubbing

Treatment of Nail Diseases

The nails are appendages of the skin which protect the ends of the fingers and toes and shield the delicate well developed tactile organs of the finger tips. They consist of water, keratin, cystine, cholesterol and mineral salts. Cholesterol maintains their elasticity and deficiency of it causes them to become brittle and dry and to break easily.

The nails grow continuously throughout the life of the individual, the matrix being the source of the nail plate. Destruction of matrix results in permanent loss of the nail and damage to matrix produces a disfigured and misshapen nail. When the matrix is exposed to prolonged irritation, subungual hyperkeratosis or hypertrophy of the nail or both may result. The nail grows from matrix to free border in five to six months, growth being more rapid in the fingers than in the toes.

The nails may be affected by cutaneous or systemic diseases. Congenital abnormalities, neoplasms, infections and dystrophic changes are observed.

CONGENITAL ABNORMALITIES

Anonychia—Congenital absence of the nail is an extremely rare condition. It may occur independently or in association with other abnormalities such as *congenital ichthyosis* (p. 3152). Supernumerary digits are usually provided with imperfectly developed nails or the nails may be completely absent.

Micronychia—Abnormally small nails occur as congenital malformations of unknown importance

Macronychia—Macronychia is a condition of abnormally large nails often associated with *clubbing of the fingers* (p 2064)

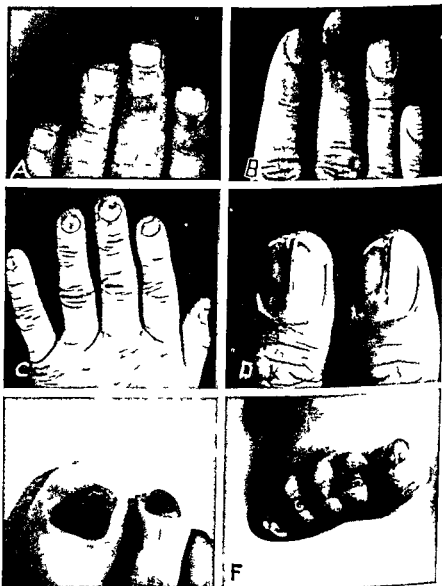


Fig 1008—A Leukonychia totalis (Stubenbord) B Leukonychia striata (Stelwagon) C Spoon nails (Stelwagon) D Splitting of nails E Dystrophy of nails following pneumonia F Pachyonychia congenita *

Pachyonychia—Extreme thickening of the nails of the fingers and toes occurs as a rare congenital abnormality. The nail is not only hardened but usually discolored and longitudinally striated. Congenital pachyonychia is usually associated with other developmental defects of the ectoderm (p

* Andrews Diseases of the Skin

3150) such as leukoplakia of the tongue and buccal mucosa and keratosis pilaris and keratoderma of the palms and soles

TRAUMA

Hangnail—Hangnails are overhanging torn portions of the nail fold accompanied by a small superficial open wound. They are important as portals of entry for pathogenic micro organisms. They are produced by injury (which may be occupational) by improper manicuring and by exposure to strong chemicals. The hangnail should be snipped off and the wound painted with tincture of iodine and dressed aseptically.

Ingrown Nail—The ingrown nail affects most commonly the great toe. An inflammatory condition of the soft tissues is produced by the abnormal

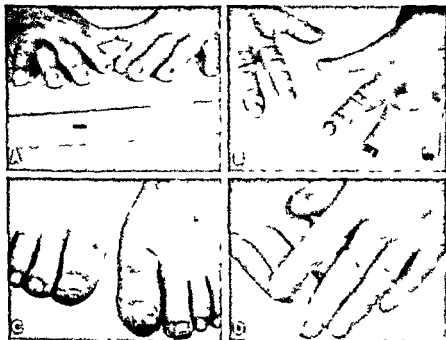


Fig 1000—A Paronychia (moniliasis) B Moniliasis C Onychomycosis D Psoriasis of nails

inward growth of the nail which results usually from too close clipping of the nail and the wearing of tight shoes. The inflammation is acute and exquisitely painful. Secondary pyogenic infection and *granuloma pyogenicum* often follow. The treatment is discussed in the section on *Minor Surgery* (p 3909).

Subungual Hematoma—Subungual hematoma is a small localized collection of blood in the nail bed usually brought on by trauma. It is observed at times in the blood dyscrasias and other diseases characterized by a tendency to hemorrhage. Where the hemorrhage is extensive enough it may cause a separation of the nail from its bed and lead to exfoliation.

The most successful therapeutic procedure is to drill a hole in the nail and evacuate the blood. This must be done soon after the onset of the condition while the blood is still fluid.

NEOPLASMS

New growths in the region of the nail are common. *Exostoses* of the phalanges about the toes are usually covered by a corn which cannot be cured until the exostosis is removed.

Glomus Tumors—The rare glomus tumor (p 3207) occurs most frequently in the nail bed and is characterized by its agonizing *painfulness*. It may encroach upon the nail bed when it becomes visible through the nail plate as a *bluish mark*. The lesion requires surgical removal.

Melanocarcinomas—Melanocarcinoma (p 3225) is a rare condition seen more often about the toenails than the fingernails. It may arise from the tissues of the matrix or the periungual fold and produces a blackish eroded or ulcerated mass which has been called inappropriately melanotic whitlow. The malignancy of the condition calls for amputation and post-operative roentgen therapy.

Epitheliomas—Epithelioma of the squamous cell type may develop about the nail especially as a consequence of degeneration of a keratosis (senile arsenical or due to radiodermatitis). *Treatment* requires combined surgery and roentgen exposure.

Fibromas—Periungual and subungual fibromas may occur independently or in association with *adenoma sebaceum* (p 3148).

Angiomas—Simple angioma (p 3200) consisting of dilated and hypertrophied capillaries may be seen as a flat red mark about the nail or in the nail bed.

PIGMENTATION

Discoloration of the nails ranging from yellow to brown to black occurs in the various forms of onychomycosis.

Workers with dyes and other chemicals often show pigmentation of the fingernails due to these substances. A bluish gray discoloration of the nail bed is a frequent concomitant of *argyria* (p 758) and may be the first visible manifestation of this metallic deposition. A brownish pigmentation of the nail bed has been noted rarely as an evidence of a drug eruption (p 3335) due to *phenolphthalein*.

Nicotine stains the nails in cigarette smokers. Topical medicaments applied to the skin may discolor the nails especially potassium permanganate, gentian violet, chrysarobin, strong sulfur solutions (Vlemminkx's solution) and resorcin.

INFECTIONS

Paronychia—Paronychia (p 3909) is an acute or chronic infection of the periungual tissues caused by the staphylococcus. In some instances fungi produce a similar condition. In the neglected pyogenic infections the process penetrates the nail bed and causes a loosening and temporary loss of the nail (Fig 1009).

Granuloma Pyogenicum—Granuloma pyogenicum may occur in the nail fold or extend out from the subungual space. It is a sequence of pyogenic infection and may be seen on the digits after paronychia and on the great toe along with ingrown toenail. It has been mistaken for melanocarcinoma (p 3225).

Tuberculosis—Tuberculous lesions about the nails are rare but it is possible for a *tuberculous chancre* (p 3259) to develop in the soft peri-

ungual tissues The *verruca necrogenica* or postmortem tubercle (p 3259) and *tuberculosis verrucosa cutis* (p 3259) also extend to the nail from the terminal phalanx

Syphilis—The syphilitic chancre (p 3278) may appear in the periungual tissues in physicians dentists midwives nurses and others It may resemble a paronychia so that incision and drainage are practiced and pyogenic infection is added to the original condition The indolence of the lesion its failure to respond to usually successful therapeutic procedures the absence of fluid pus and the painless disproportionately enlarged epitrochlear or axillary lymph nodes should arouse suspicion of syphilis A darkfield examination (p 45) reveals the spirochete at a time when the serologic tests are still negative (p 336)

Anaerobiasis—Anaerobic infection of the periungual tissues with *fuso spirillary organisms* is a rare occurrence following trauma especially a human bite The lesion is indolent crusted painful and moist and smears reveal the Vincent's spirillae and fusiform bacilli

Treatment consists of the local application of oxidizing agents such as diluted hydrogen peroxide (25 per cent strength) hot potassium permanganate soaks (1:4000) or a 10 per cent solution of arsphenamine in glycerine Zinc peroxide paste may be used as a continuous dressing In refractory cases intramuscular injections of penicillin are advised until the lesion has healed

Verruca—*Verruca vulgaris* (p 3288) may appear in the lateral folds of the nail or in the subungual space At times plantar warts extend into the proximity of the toenails Warts about the nails present a therapeutic problem Roentgen ray therapy may be successful Electrodesiccation and curettage may be necessary but require the cutting away of a section of the nail to obtain satisfactory exposure of the lesion

Onychomycosis—Onychomycosis due to trichophytons and epidermophytons (p 3293) is rarely accompanied by paronychia The nails of the toes are involved more often than those of the fingers They appear thickened dry discolored lusterless and irregular Subungual hyperkeratosis and partial separation of the nail from its bed may also be present

Favus may affect the nails but lesions of the scalp or skin are always coexistent The nails are thickened yellowish and distorted and there are subungual accumulations of horny material

Monilia—Chronic paronychia due to monilia and other yeasts is a not uncommon condition It is most often observed in individuals who are obliged to immerse their hands in water for considerable periods of time The nail is almost invariably diseased and becomes discolored opaque and separated from its bed (Fig 1009)

NAIL CHANGES IN CUTANEOUS DISEASES

Abnormalities of the nails occur with cutaneous diseases as a result of direct extension or the actual incidence of the characteristic cutaneous lesions in the nail bed In the former instance the changes are likely to be nonspecific while in the latter they may be specific

Psoriasis—The cutaneous disorders that affect the nails most commonly are *onychomycosis* and *psoriasis* In the latter condition the nail lesion may be the sole manifestation or it may merely be a portion of a wide-

spread disturbance The changes in *psoriasis* (p 3414) assume two varied manifestations The commonest is a punctate pitting of the nail plates with the appearance of transverse ridges This is usually accompanied by extensive thickening of the nail bed and elevation of the nail plate A second characteristic form is the appearance of scaling papules under the nail plate which later becomes distorted

When the *psoriasis* of the nails is accompanied by other cutaneous manifestations the *diagnosis* offers no difficulty The differentiation between *psoriasis* and a fungous infection may be aided by scrapings and cultural studies for fungi

Contact Dermatitis.—*Dermatitis venenata* (p 3330) of the hands and fingers may spread to the nails and cause secondary pyogenic paronychia with temporary loss of the nail The nails may show evidence of trophic disturbance becoming distorted ridged lusterless and brittle In chronic eczematous conditions the nails become atrophic or hypertrophic Sensitivity to nail polish rarely causes changes in the nails or perungual tissues most commonly bringing on *dermatitis venenata* in such remote situations as the eyelids chin and neck

Exfoliative Dermatitis.—Exfoliative dermatitis (p 3383) is often accompanied by deformation of the nails but the most typical alteration is a complete shedding of the nails This is most often seen in drug sensitivity (p 3812) in which regrowth of the nails and hair takes place after the disease is controlled

Epidermolysis Bullosa.—In *epidermolysis bullosa* (p 3151) of the dystrophic variety there is often total loss of the nails with atrophy of the matrix so that there is never regrowth

Alopecia Areata.—*Alopecia areata* (p 3445) may be accompanied by a variety of deformities in the nails In the severe instances exfoliation occurs

Radiodermatitis.—*Radiodermatitis* (p 3179) of the fingers may be accompanied by atrophic changes in the nails This develops in those whose occupation exposes them to roentgen rays or radium or in patients who have received excessive dosage for treatment of a local disease

DYSTROPHIES

Dystrophies of the nail accompany local and systemic disorders Trophic changes in the cutaneous diseases are discussed elsewhere (p 3455)

Various types of atrophy also accompany trophic disturbances of the extremities such as injuries to the peripheral nerves impairment of the local circulation as in arteriosclerosis or the less common forms of peripheral vascular disease in Raynaud's disease and scleroderma

Again the nail dystrophy may be part of a more widespread metabolic disturbance such as occurs in primary anemias pulmonary tuberculosis the atrophic types of arthropathy endocrine dysfunction vitamin deficiency states and the severe and protracted febrile disorders

Onychauxis and Onychogryphosis.—*Onychauxis* is a hypertrophy of the nail most frequently of the great toe It may affect several toes and more rarely the fingernails The nail is thickened elongated distorted and discolored Beneath the nail there is an accumulation of dry

horny material (*subungual hyperkeratosis*) When the nail becomes exceedingly elongated curved and clawlike the condition is called *onycho gryphosis* This abnormality has been observed in neurological conditions with associated trophic disturbances but commonly results from undue pressure and bad hygiene It has occasionally been encountered in endocrine dysfunction notably *acromegaly* (p 1156)

Onychomadesis—*Onychomadesis* denotes shedding of the nail beginning at the matrix and progressing to the free margin Eventually the nail becomes completely separated and is exfoliated but it practically always regrows It may affect one or more nails and all the nails of the fingers and toes may be cast off

Onychomadesis may be the result of a local disease such as *paronychia onychomycosis* or *subungual hematoma* As a *polyungual* condition it usually follows a severe systemic disease such as scarlet fever exfoliative dermatitis (p 3383) total or universal *alopecia areata* (p 3179) or *epidermolysis bullosa hereditaria* (p 3151) It is also observed in certain occupations especially where cement and strong chemicals are handled

Onycholysis—*Onycholysis* is a partial separation of the nail from its bed without actual exfoliation The process begins at the free margin and progresses backwards to the matrix It is often seen in industrial workers whose hands are immersed in water for long periods especially when soap and chemicals are also present Bartenders dishwashers laundresses and others are often affected It may be a consequence of *subungual hyperkeratosis* which lifts the nail from its bed as in *onychomycosis* and *psoriasis* (p 3414)

Onychorrhexis—*Onychorrhexis* is a common condition characterized by longitudinal ridging of the nails at times accompanied by fissuring The nail becomes thinned and breaks off easily at its free margin It is seen in elderly persons and workers exposed to strong alkalis and acids

Transverse Lines (Beau)—*Transverse lines* (*Beau's lines*) are linear depressions in the nails which are first noted at the proximal part of the nail and progress steadily towards its free margin as the nail grows They are removed when the nail is clipped and do not reappear unless the causative condition operates again Since the full growth of the nail takes place in five to six months it requires this length of time before the line disappears

The lines are caused by some temporary nutritional interference with nail production by the matrix They occur most often after a severe illness infectious disease mental shock or pregnancy

Onychatrophia and Onychophagia—*Onychatrophia* signifies the state in which the nails are thin poorly developed lusterless and broken off irregularly at the free margin In the severer examples most of the nail may have separated away only a few fragments remaining at the base Local disease such as fungous and pyogenic infections cause atrophy of the nail It is not uncommon in industrial workers as a result of trauma or of strong chemicals It may be associated with endocrine dysfunction such as *hyperthyroidism* and *hypopituitarism*

The ends of the nails may be broken off and worn down by prolonged scratching The nails are shortened in those who have the uncontrollable habit of nail biting (*onychophagia*)

Fragilitas Unguium—*Fragilitas unguium* is a condition in which the nails become dry and brittle and crack and break off readily. This is most frequent in women and is ascribed to the continued use of nail enamels, enamel removers and cuticle removers which have a deleterious effect upon the nail plate.

Koilonychia—*Koilonychia* (spoon nails) is an uncommon abnormality in which the nail plate loses its normal convexity and actually becomes concave. One or more fingernails may be affected. The nails are usually quite normal apart from their crateriform shape. Although it has been noted in association with syphilis, cachexia, anemia and other diseases, its fundamental cause is unknown. It is said to occur with a fair degree of frequency in workers engaged in the manufacture of glass.

Leukonychia—*Leukonychia* is a very common condition in which one or more nails display white spots or lines, usually arranged transversely. Rarely the entire nail may become white. The white markings advance as the nail grows and disappear at the free margin.

The cause is not known but trauma from the manicure and unknown constitutional abnormalities have been blamed. Total leukonychia in which all the nails are completely whitened is rare and is said to be congenital and hereditary.

Clubbing (*Hypertrophic Pulmonary Osteoarthropathy*)—Clubbing of the fingers is often accompanied by deformation of the nails. The latter are normal except that they are curved more sharply than normal in both transverse and longitudinal directions. The free margin arches acutely downward. Chronic cardiac disease, especially congenital heart malformations, emphysema, bronchiectasis and pulmonary tuberculosis are most often responsible (p. 2064).

In *chronic arthritis* the nails are dry and brittle, deformed, thickened and occasionally exfoliate.

TREATMENT OF NAIL DISEASES

The treatment of the underlying systemic or cutaneous disease, the avoidance of occupational traumas, the improvement of nail hygiene, the correction of foot deformities, the wearing of well fitted shoes and careful or less frequent manicure are all important in the handling of the above conditions. The management of the infections and neoplastic conditions has been discussed in the sections to which reference is made.

A valuable form of therapy is by roentgen rays. This is of particular benefit in fungus infections, chronic paronychia, psoriasis, dermatitis venenata and verruca vulgaris. Naturally the dosage must be properly controlled and where the disease is recurrent the patient must be warned against the dangers of repetition or overdosage.

CHAPTER 153

DISTURBANCES OF THE SWEAT GLANDS

Physiology and Pharmacology of Sweat

Clinical Manifestations of Disturbances of the Sweat Glands

Hyperidrosis

Anidrosis

Bromidrosis

Chromidrosis

Hematidrosis

Sudamen (*Miliaria Crystallina*) (p 3169)

Miliaria Rubra (Prickly Heat) (p 3171)

Hidradenitis Suppurative Axillaris (p 3253)

Syringocystoma (p 3209)

Hydrocystoma

Fox Fordyce Disease

Cranulosis Rubra Nasi

PHYSIOLOGY AND PHARMACOLOGY OF SWEAT

PERSPIRATION is a secretion of the sweat glands. The sweat glands are found over almost the entire cutaneous surface. It has been estimated that the total number of them approximates two million.

Daily Secretion—The amount of sweat formed by the glands varies greatly being influenced by atmospheric conditions as well as the physical and psychological state of the individual. In the normal person at rest the average quantity for twenty-four hours varies between 500 and 600 cc. After muscular exercise or under the influence of high external temperature the amount may be increased to as much as 2500 cc daily.

Chemistry of Sweat—The specific gravity of sweat is low, normally measuring about 1.004. The normal reaction is distinctly acid, the average reaction being pH 5.65, although it may at times become alkaline or neutral. The main inorganic constituent of the sweat is *sodium chloride*. *Urea* occurs in amounts that approximate the concentration in the blood. In azotemia (p 2276) the crystals deposit on the skin as *urea snow*. The excretion of mineral by sweat may be sufficient to produce manifestations of chloride deficiency in those who perspire profusely. Workers in steel foundries, stokers and cooks develop violent muscular cramps when large quantities of plain water are drunk. These symptoms are prevented by the addition of sodium chloride to drinking water.

Function of Sweat—Sweat maintains the moisture of the skin and prevents the scaling and cracking that results from dryness. It is an important factor in the regulation of body temperature. The dry, burning skin characterizes temperature elevation while diaphoresis accompanies or follows defervescence.

The excretory function of sweat, particularly referable to water balance, is often underestimated. The moisture of the skin may evaporate without appreciable or visible evidence and is then known as *insensible perspiration*. The latter accounts for a greater water loss per diem than

occurs through urine and feces combined. This virtue is metabolically acceptable but often constitutes an esthetic handicap hence the wide use of cosmetic anidrotics (p 3142)

Therapeutic Diaphoresis—Attempts to utilize the skin as an excretory organ in renal insufficiency by effecting profuse diaphoresis through physical modalities packs and drugs are usually defeated by the exhaustion attendant upon the vigor of the measures and the cachexia of the patient.

Innervation of the Sweat Glands—The innervation of the sweat glands is through the involuntary nervous system. The cholinergic or vagus mechanism is the principal secretory structure since physostigmine and pilocarpine promote diaphoresis while atropine dries the skin. It is possible that secretory nerves also arise in the adrenergic division since sweating is associated with the injection of epinephrine and occurs in many diseases such as hyperthyroidism characterized by evidences of increased activity in this portion of the involuntary nervous system.

In addition to the peripheral nervous mechanism there is evidence to show that *sweat centers* exist in the cord. This mechanism is probably stimulated in the sweating crises of *tabes dorsalis* (p 1464). There is also a general regulating center in the medulla which responds to emotional episodes and to profound vascular disturbances especially those seen in *shock* (p 928).

CLINICAL MANIFESTATIONS OF DISTURBANCES OF THE SWEAT GLANDS

HYPERIDROSIS (INCREASED SWEATING)

Increase in sweat gland activity may be local or generalized.

Local Hyperidrosis—Local hyperidrosis occurs most commonly in the *hands* and *feet*. In many instances this is a constitutional or psychogenic disturbance most commonly observed in adolescents and adults who exhibit other evidences of neurogenic and emotional instability. The increase in sweat is often independent of the temperature of the part. Many young persons exhibit the combination of cold extremities with increased sweat. Localized sweating may be exhibited in other locations such as the brow, the tip of the nose, a single finger, the hypothenar eminence, the axillae, under the breasts, and between the buttocks or thighs.

More rarely localized sweating occurs as the result of isolated nerve injury. Thus in *aneurysms* and *mediastinal tumors* which impinge upon the cervical sympathetic there may be unilateral sweating of the face together with the manifestations of Horner's syndrome (enophthalmos, miosis). In *tabetic crises* the girdle area may be delineated by localized sweating.

Treatment—Localized sweating requires prophylactic therapy to prevent intertrigo and secondary infection producing dermatophytosis (p 3293) and the pyodermas (p 3248). Ordinarily this can be accomplished by frequent bathing, thorough drying, sponging with alcohol and the liberal use of talcum powder. Under special circumstances such as profuse sweating of the feet, it is wise to use an antiseptic powder (p 3137).

For sweating under the arms and in the folds and creases of the skin the cosmetic anidrotics (p 3142) are employed in solution or lotion. Preparations of aluminum chloride (p 3113) are most satisfactory but their

continued use may produce irritation Formaldehyde mercury and lead washes are potentially dangerous and should be avoided Ointments and creams are usually unpleasant but some of the aluminum preparations commercially obtainable are not at all objectionable Frequent bathing or washing thorough drying and the free use of unscented talcum powder often accomplish more than the elaborate and expensive preparations

Roentgen ray exposure benefits local hyperidrosis but the expense and danger of the treatment outweigh the advantages In average safe dosage the results are temporary the doses required to produce a permanent effect being likely to produce radiodermatitis

Generalized Hyperidrosis—Generalized sweating occurs in response to a variety of stimuli There is a wide individual variation in sweating Obese patients are apt to sweat more profusely the lean ones and children perspire more freely than adults and elderly people

Physiologic and Pharmacologic Causes—People perspire freely in hot weather following or accompanying exercise when they are nervous or under emotional strain, as the result of the administration of cholinergic drugs such as physostigmine and pilocarpine accompanying nausea and vomiting and after the administration of the antipyretics or emetics such as ipecac

Pathologic Causes—Excessive sweating occurs as an incident in a variety of pathologic conditions such as shock collapse and febrile states accompanied by the swings of temperature which occur naturally in malaria and sepsis and artificially after the use of antipyretic drugs

In other conditions sweating may be a dominant or an arresting symptom Head sweating characterizes rickets (p 2850) in infancy and early childhood Generalized profuse diaphoresis occurs in hyperthyroidism (p 1197) the night sweat arouses the suspicion of an active tuberculosis (p 252) acrid sour-smelling sweat occurs in rheumatic fever (p 186) while the sweat has a musty odor in typhoid states (p 225) Cold clammy sweat arouses attention in conditions of shock and collapse (p 928) particularly during or following surgery and in coronary thrombosis (p 983)

Sweating accompanies the hot flushes of the menopause (p 2493) and is also a prominent complaint in convalescence and states of general debility

Suggested Additional Examinations—1 *Temperature record* (p 3484) low grade pyrexia in tuberculosis and rheumatic fever wide fluctuations in sepsis and malaria

2 *Estimation of basal metabolic rate* (p 3738) elevation in hyperthyroidism

3 *Radiograph of chest* (p 3741) positive findings in insidious tuberculosis

Treatment—The treatment of sweating requires the elucidation of its cause and the management of the more fundamental condition

In febrile states the patient should be thoroughly sponged dried and powdered to avoid chilling or the development of the sweat gland disturbances such as sudamina (p 3169) and miliaria rubra (prickly heat) (p 3171) It is inadvisable to use atropine and related drugs to dry the sweat since side-effects such as dilatation of the pupils and dryness of

the mouth create considerably more discomfort than the symptom under treatment. The night sweats of tuberculosis may be allayed by a warm tub or a shower at bedtime followed by an alcohol rub and powdering.

Patients and workers who perspire profusely should be encouraged to ingest sodium chloride in drinks or in capsules containing ordinary table salt.

ANIDROSIS

Complete anidrosis is an extremely rare condition. More frequently diminished sweating accompanies dermatoses and systemic disorders.

Anidrosis in the Dermatoses—Complete absence of sweating is extremely rare and is probably encountered only in *congenital ectodermal defect* (p 3150).

Diminished sweating is seen with those cutaneous conditions in which sweat glands have been damaged or destroyed. *Hypodidrosis* follows extensive atrophy or scarring from any cause; it is noted in ichthyosis, exfoliative dermatitis, granuloma fungoides and pemphigus. In these conditions the inability of the skin to dissipate body heat may cause a febrile pyrexia.

Anidrosis in Systemic Disorders—Diminished sweat gland activity is associated with *senility*, *myxedema* and *scleroderma*.

Treatment—Diminished sweating rarely necessitates treatment. Activation of diaphoresis may be induced by the application of dry heat, massage, ingestion of hot liquids and the injection of pilocarpine (p 3374). In the anidrosis of congenital ectodermal defect no treatment avails since sweat glands are absent. Such patients should avoid excessive heat, violent exercise and everything else which increases sweating.

BROMIDROSIS

Bromidrosis is a condition in which the sweat has a foul odor or becomes malodorous due to decomposition. It may or may not be accompanied by hyperidrosis and may be generalized or localized.

Generalized Bromidrosis—An unpleasant sweaty odor results most often from uncleanness and failure to bathe or change the clothes that are in direct contact with the skin. Generalized bromidrosis may also result following the ingestion of onions or garlic or after the use of odoriferous drugs such as valerian or asafoetida. Certain of the systemic diseases such as typhoid and rheumatic fever have a characteristic odor. In the typhoid state the sweat is described as musty whereas in rheumatic fever the odor is acid.

Racial differences occur in the odor of sweat and each race finds all others possessed of an offensive smell. The highly developed olfactory apparatus of the bloodhound is capable of distinguishing the personal characteristics of body odor.

Localized Bromidrosis—Localized bromidrosis is most common in the folds of the skin and on the feet. For the most part it results from warmth, moisture, uncleanness and dirty linen but it may persist despite all efforts at hygienic correction. In many instances ringworm of the feet gives rise to an unpleasant odor to the degree that the condition is often vulgarly called stink foot. It is claimed that this odor is the result of decomposition by saprophytes especially *Bacillus foetidus*.

Odors in the anogenital region most often arise from inspissated smegma under the foreskin from decomposing feces caught in the hairs of the anal region or from a vaginal discharge

Treatment—The relief of bromidrosis requires that odoriferous drugs and foods be forbidden. General hygienic measures include frequent bathing, changing of linen and the teaching of cleanliness after stool. With smelly feet the interdigital area is examined for evidences of ringworm which must be eradicated (p 3307). Under any circumstances the feet should be bathed frequently and thoroughly dried, particularly in the region between the toes. Simple application of bicarbonate of soda may be most effectual.

A useful powder consists of

| | | |
|---|------------------|---------------|
| R | Salicylic Acid | 1.2 |
| | Thymol | 0.6 |
| | Zinc Oxide | |
| | Boric Acid | |
| | Talcum | ss qs ad 60.0 |
| | Make a powder | |
| | Sig External use | |

In extreme instances the feet may be bathed with 1:1000 potassium permanganate. The use of perforated shoes is advisable. In desperation roentgen ray treatment may be required and should be followed by signal success.

These measures failing the afflicted may then resort to sachets, toilet waters or perfumes.

CHROMIDROSIS

Chromidrosis is a rare condition in which the sweat is colored when secreted or becomes tinged after exposure to the air. The colors reported include yellow, red, green, blue and black. The discoloration is most commonly seen in sweat secreted about the lower eyelids and the adjacent cheeks but may be present in other areas of the body.

The disorder is very rare and has been observed chiefly in neurasthenic females. It has been ascribed variously to the excretion of metallic substances (such as copper, phosphorus and iron) or to metabolites such as indican and hemoglobin derivatives. Some instances are undoubtedly due to the action on the sweat of chromogenic bacteria or fungi (p 3300).

Treatment requires the discontinuance of drugs which may be responsible. Local measures are instituted against possible bacterial or fungous agents (p 3307).

HEMATIDROSIS

Hematidrosis is a rare condition in which blood is exuded with the sweat. It may occur in blood dyscrasias or hemophilia and may also represent a form of vicarious menstruation.

Most examples observed in psychotic females with supernatural religious fantasies may well be self-induced.

SUDAMEN (MILIARIA CRYSTALLINA)

MILIARIA RUBRA (PRICKLY HEAT)

See p 3171

HIDRADENITIS SUPPURATIVE AXILLARIS

See p 3253

SYRINGOCYSTOMA

See p 3209

HYDROCYSTOMA

Hydrocystoma is a chronic cystic disorder of the ducts of the sweat glands of the face. The disease is most frequently observed in elderly women who work as cooks or laundresses. Increased sweating and exposure to steam predispose to the condition.

The Eruption—The eruption is confined to the face, particularly the nose adjacent cheeks and the forehead. It consists of multiple discrete faintly yellowish or white pinhead to pea sized vesicles. There may be only a few lesions or several dozen. No evidence of inflammation is present. The vesicles are only slightly elevated and are felt deeply within the skin. They do not rupture but when pierced there is a slight flow of colorless sweat. The condition is at its worst in the summer or when sweating is excessive.

Diagnosis—The absence of itching and inflammation differentiates hydrocystoma from *dermatitis venenata*. Puncture of the lesions reveals their cystic nature and excludes *benign cystic epithelioma adenoma sebaceum* and *milium*.

Treatment—The patient is instructed as to the cause and advised to avoid exposure to excessive heat and steam. The individual cysts may be pierced and evacuated. Exposure to roentgen ray is usually curative.

FOX FORDYCE DISEASE

Fox Fordyce disease is a rare condition affecting the apocrine sweat glands of the axilla, the pubic area and the region about the nipples. It occurs almost exclusively in the female, usually between the ages of twenty and thirty. Its exact causation is unknown but it has been attributed to endocrine disturbances, alterations in the chemistry of the sweat and toxic factors.

The Eruption—The eruption consists of a profusion of closely crowded lentil sized or smaller rounded slightly red papules. The hair may be absent from the affected sites as a consequence of scratching. The itching is indescribably intense, persistent and intractable.

Diagnosis—The strict confinement of the condition to the apocrine gland structures differentiates this disease readily from *neurodermatitis* and *lichen planus*.

Treatment—Therapy is not particularly successful; the best results having been achieved with exposure to roentgen rays. Antipruritic topical applications (p 3136) may be of slight value.

GRANULOSIS RUBRA NASI

Granulosis rubra nasi is a rare affection seen in children from infancy up to about 16 years of age. It may be due to local circulatory deficiency.

or disturbance of the involuntary nervous system. The condition tends to spontaneous cure as the patient approaches adulthood.

The disease is confined to the nose, which is cold and moist and perpetually covered with droplets of sweat. There is a general redness of the



Fig. 1010.—Fordyce's disease of the right ala. Before an epulating dose of filtered x rays, which caused permanent regression of the disease.

nose and discrete dull red macules and papules are present. Occasionally hydrocystomas are noted.

Spontaneous recovery can be anticipated. Therapy should be simple, including attention to the general health and the local application of talcum powder containing 1 per cent salicylic acid.

Andrews: Diseases of the Skin

MILIARIA RUBRA (PRICKLY HEAT)

See p 3171

HIDRADENITIS SUPPURATIVE AXILLARIS

See p 3253

SYRINGOCYSTOMA

See p 3209

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SECTION XXI

THE TECHNIQS OF PHYSICAL DIAGNOSIS

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Sufferers from cancerophobia may one day turn up with genuine malignancy

The physician is safer in the assumption that the history as given is authentic and accurate. He should continue to believe the narrative until his credulity is strained to the breaking point.

THE PRESENT ILLNESS

The Patient's Narrative—The history in private practice should be initiated by a full statement of the *present complaint*. The present illness is the reason for the consultation. The patient has come to narrate his story. He will often be impatient if he is first quizzed concerning what are to him irrelevant matters such as the cause of the death of his parents or the number of aunts and uncles with high blood pressure.

The patient may be asked: What is your complaint? or What is your difficulty? The answer often constitutes the entire content of the present illness. It is wise not to interrupt the narrative. When possible the story should be heard without suggestion or interference.

At other times the history is disclosed piecemeal during the conduct of the physical examination or at subsequent visits. The complexity of the story of a prolonged or recurrent disturbance may be such that it is best reviewed after the determination of the physical status and in the light of the positive findings. In these latter instances it is particularly important to avoid writing the chart until the entire situation is somewhat clarified.

Definition of Terms—When the narrative has been completed by skillful questioning the historian fixes the facts more accurately and obtains additional data. The first necessity is a common agreement on terminology. The patient may use the word *feverish* to mean flushed or warm. He may state that he has a *pain* in his stomach and mean the epigastrium, the groin, or any portion of the abdomen adjacent thereto. With the complaint of pain the patient should be asked to point to the involved region.

The physician will of course use tact and his own judgment regarding the terminology he uses when questioning patients. Generally however the vernacular should be employed rather than adhering in a dignified fashion to technical terms. Thus a patient may deny he has had a venereal disease but readily admit to having had *clap*.

Whatever the present complaint there should be no doubt at the end of the interview that both the patient and physician have a common understanding concerning the terms employed.

Time Relationships—The approximate *onset* and the *duration* of the present complaint are of vital significance in diagnosis. When the patient seems to be in doubt it may be helpful to ask: When were you last well? or relate the time to outstanding dates such as New Year's Day, Easter, Thanksgiving, or Christmas. Again the time relationship may be fixed relative to menses, birthdays, anniversaries, or holidays.

Attendant Circumstances—The attendant circumstances should be delineated. The relationship of abdominal pain to *meals*, *defecation*, or the *menstrual period*; of precordial pain to *exertion*; or of skeletal pain to *exercise* may prove to be important points in the diagnosis.

Effects of Previous Therapy—The results of preventive measures and attempts at relief should be reviewed. This information gives a clue to the

THE PHYSICIAN AS A HISTORIAN

To meet varying circumstances the physician employs many tactics. At times he need only lend an attentive ear while details are poured forth again it may be necessary to elicit information by skilful questioning, acting the role of an historical midwife.

The *attitude of the physician* during recital of the history should be one of sympathy and concern. The presence of a third person may inhibit a free flow of information. The attempt to write the history as it is given interrupts conversation and detracts from full concentration. It is wiser to digest and mentally edit the story before committing it to the permanent record. This method has the disadvantage that facts may be forgotten or colored by the historian. With practice however, a reasonable degree of accuracy is developed.

The physician must retain complete composure in the face of startling revelations. He must avoid moral judgments of other human beings and maintain the sanctity of the professional relationship not only to the patient but also to others whose lives are entwined or tangential. It is often helpful for the physician to reconstruct situations that the patient describes. For this purpose an imaginative mind adequately checked is of great value. The robust physician finds difficulty in appreciating the illness and suffering that is more clearly envisaged by the more sensitive or even neurotic practitioner.

The physician should not assume the attitude of a district attorney even when statements seem to be inaccurate, exaggerated or wholly fallacious. The patient must be prevented from recognizing any skepticism on the part of the inquirer even though the clinical history is to be interpreted according to the historian's estimation of the accuracy of the recitalist. One individual insensitive to pain may suffer so ominous a disturbance as a coronary thrombosis with little subjective distress. Another may be a confirmed stoic and understate symptomatology as a pose through fear of examination or because of dread of undesirable sequelae. A child may fail to report a pain in the ear lest he be forbidden to go swimming. A pathologically modest woman might not mention a vaginal discharge to avoid pelvic examination. An employee might deny tuberculosis or known syphilis lest his job be forfeited. Patients in a phase of negativism not only refuse to volunteer subjective complaints but may even deny their presence when questioned.

Much more common is the *overstatement* and *exaggeration* of symptoms. The practitioner has his ears assailed by the statement that the head is bursting, the pain agonizing and the sufferer never slept a wink the entire night. Correct evaluation of an overly picturesque narration places an onus and responsibility upon the physician. The patient resents the justifiable doubt and labels the physician as unsympathetic or lacking in understanding. He seeks other ears into which to pour the complaints and the physician suffers from the error of being correct.

When the gravity of the symptoms has been underestimated the outcome may be grievous for the patient and physician alike. A neurotic patient of an alarmist character may for example actually have a gangrenous appendix, unrecognized because of his physician's incredulity.

The Pattern of the Way of Life—A *chronological* recital of the normal activities of the average day is the best method of obtaining a complete personal history. From this the practitioner obtains a clear conception of personality and character traits and the occupational and environmental factors.

The patient should recount exactly what time he arises, what is eaten for breakfast, when the bowels move and the character, odor and color of the bowel movement, the number of times urination occurs during the day, the nature of the morning activities, the usual luncheon, the nature of the afternoon activities, the character of the work done, the possible occupational hazards, the amount of emotional, mental and physical strain involved, the time of completion of the day's activities, the ingredients of the evening meal, the amount of alcohol used, the number of cigarettes, cigars or pipes smoked, the regular uses of unprescribed drugs, such as laxatives, coal tar preparations, sedatives, hypnotics, narcotics, pick me ups, and the like, the nature of the recreational activities in the evening, the time the patient goes to bed, the nature of the sleep, the nature of the awakening, the sexual habits, the frequency of nocturnal urination, and finally, when indicated, the patient's general outlook toward his life, his ambitions and interests, his frustrations, difficulties, hardships and disappointments.

In Children—With children and adolescents the parents should be interrogated concerning the gestation history, the type of labor, birth trauma, and the chronology of developmental processes.

Boys and girls of school and college age should discuss their academic progress, the type of grades, the approach of expected examinations, and the outlook as to the future, as well as their interest in extra curricular activities—athletic, cultural and amorous.

In the Female—Of the women patients it is important to learn the age of the onset of *menses*, the regularity of the period, its duration and quantity, the amount of pain experienced, the character of the flow, the general constitutional symptoms that precede, accompany or follow the actual period, and the symptoms that occur during the middle period or ovulation time.

During the years of active sex life the patient is questioned concerning the number of *miscarriages* and *stillbirths* (whether spontaneous, suggesting syphilis or induced, suggesting pelvic disease from the ministrations of an illegal practitioner), the number of pregnancies, the general health during the *period of childbearing*, the *presence of complications*, particularly those suggesting kidney damage and hypertension, the character of the labor, and local trauma incident to childbirth.

Of the nulliparous female inquiry should be made concerning the possible causes of *sterility*, whether due to the practice of contraception or despite earnest effort at impregnation. When indicated by the trend of the history, unmarried women should be quizzed concerning sexual exposure, and married women relative to sexual habits. In the case of older women the date of the *menopause* and its attendant difficulties should be noted.

In the Male—In men *occupational hazards* are investigated. Exposure to undue risks may be elicited from the history. In most unionized industries workers are familiar with industrial poisonings and hazards. They

diagnosis It may eliminate unsuccessful measures from the therapeutic program, and suggest the incorporation of the successful efforts Thus the application of heat should be continued if the patient stated that relief was obtained from the electric pad It would be futile to suggest the use of a coal tar preparation if pain persisted despite the ingestion of large amounts of some popular brand of analgesic drug

Tempo of Illness—The tempo and the intensity of the symptoms throw light on the diagnosis and especially the prognosis It is important to know whether pain is *increasing* or *decreasing* whether in infection there is a *rise* or *fall* of the temperature curve It is more important to note sudden changes in weight or physiologic habits than simply that a patient has always deviated from the norm

FAMILY HISTORY

The family history need cover only the most pertinent data It is important to know the *marital state* the *number of children* *miscarriages* and *stillbirths* and the *general state of health* of the family group and marital partner

This information is of the greatest importance in familial or hereditary disease such as *hemophilia* *allergy* and certain *neurological conditions* It is also significant in constitutional diseases such as *arteriosclerosis* *diabetes* and *nephritis* It is of the highest importance in infectious diseases which like *syphilis* are transmissible or in *tuberculosis* where there is a diminished resistance in certain racial and familial groups

Here as elsewhere only *positive findings* are recorded It is unnecessary to append lists of absent and normal events and findings followed by the dubious term *negative* The methodical practitioner may assume that absence of a notation in his record indicates normalcy and not an omission

PERSONAL HISTORY

The *personal history* while relatively insignificant in hospital records is of the utmost importance in private practice Many patients are ignorant of the fundamentals of personal hygiene Symptomatic complaints frequently can be clarified and relieved by the discovery, elimination or correction of the culpable factor in the daily routine It requires no great diagnostic skill to decide that a housewife is suffering from an *exhaustion syndrome* (manifested by headache asthenia fatigability or insomnia) if she states that from the preparation of breakfast at 6 30 A M she works constantly each day in the year until her supper dishes are washed and put away at 9 30 P M

Many obscure attacks of *recurrent headache* may be due to the habit of ransacking the ice box at bed time or to overindulgence in alcohol or chocolate, or other foods and beverages particularly in the case of those with an idiosyncrasy or allergy Poorly balanced diets may give rise to *vitamin deficiencies* which may be accurately diagnosed and treated only when the complete eating habits are investigated Many *gastric* and *intestinal disorders* and particularly those associated with nervousness and irritability result from the use of excessive amounts of coffee or tea

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may be asked directly whether their own symptoms are similar to those of other workers in their field.

Men may be frankly questioned concerning sexual history sexual habits and venereal disease

In Functional or Psychosomatic Disturbances—When there is a suspected functional disturbance a functional overtone to organic disease or a clear psychiatric component to a clinical problem it is necessary to learn the patient's attitude toward his illness and the support and solace he receives from religion reading music or hobbies of one type or another

Investigation of the family relationship may disclose conflict between husband and wife extramarital philanderings schisms between parent and child sister and brother, or relatives closely linked in the family circle These facts are of paramount importance not only in evaluating the symptoms but also in arranging the therapeutic regimen

Pattern of Life and the Therapeutic Program—Social and economic considerations greatly modify medical care In ordinary practice expensive diagnostic investigation cannot be ordered unless there is real suspicion of organic disease or the symptoms do not respond to usual treatment In the interest of economy pure science must yield and a probative therapeutic regimen should be inaugurated

An overworked mother with the complicated demands of a large household cannot always be expected to follow a special diet A trainman cannot very well follow a regimen requiring frequent feedings A day laborer cannot find a sedentary occupation when his vessels begin to sclerose A travelling salesman should not be expected to lead a regulated life with regard to hours of sleep food and bowel habits

It is often better to compromise and accomplish something less than the ideal rather than offer a rigid program which cannot possibly be followed for more than a few days at best

To be successful a therapeutic regimen must be 'tailor made' conforming to the life pattern of the patient It is wiser to fit the program to the patient than to alter the patient's life to suit his treatment schedule

Economic Status—In wage earners male or female the economic status should be discovered in addition to occupational hazards If the patient is employed inquiry should be made concerning salary and the security of the position The small business man the owner or director of an enterprise should state the problems and difficulties in the economics and operation of his venture

The Diary History—Complicated histories may be clarified by requesting the patient to keep a diary making entries cataloguing the activities habits food intake exposures and contacts

The 'diary' method of history is invaluable in dealing with chronic or recurrent symptoms such as *headache* It may be impossible for the patient to give all of the required data in the recital of the personal history When there is suspicion of sensitivity to drugs chemicals cosmetics or foods the patient may not be able to state accurately whether an eruption or a headache or an attack of diarrhea followed exposure to a suspected substance The diary may reveal some common factor that recurs in association with the symptoms thus clarifying obscure allergic dermatologic or gastro intestinal disorders

PAST HISTORY

The past history is the last portion of the anamnesis. It is a relatively brief recital of those ailments, operations or accidents from which the patient has suffered. It is quite unnecessary to write down all the "negative data." *Terms used by the patient should be defined.* Thus a patient may understand conditions far from specific in the use of words such as "rheumatism," "malaria," and "kidney trouble."

It is essential to note the *severity* and *duration* of the past illnesses as well as the complications. This information is useful in determining the reaction of the patient to disease and may be of help in prognosis. Infections with possible late sequels such as the exanthems, rheumatic manifestations, pertussis, diphtheria, syphilis and tuberculosis must be minutely described. *Idiosyncrasy* to drugs or biologicals is noted. Sensitization to horse serum from injection of tetanus antitoxin or the like must be imprinted on the practitioner's mind as well as on the record. Note should be made concerning immunization specifically with relation to smallpox, typhoid fever, tetanus, diphtheria, pertussis and scarlet fever.

The knowledge of *previous attacks* of exanthems or immunization will often assist in the diagnosis by exclusion of an acute febrile disease. Abdominal diagnosis is made considerably simpler if it is known that the appendix has been previously removed. *Veneral infection* must be specifically acknowledged or denied. Past residence and military service in tropical countries suggest parasitic or unusual infectious diseases. Questioning should in most instances employ vernacular rather than pedantic language.

Concerning *surgical procedures* the practitioner should learn when these were done, the indications for the operation and the name of the surgeon. The patient's experiences will serve as a follow up and give some insight into the skill and practices of the operating surgeons in the environs. Single statements are of little value but it will not be long before a fairly large experience is accumulated.

CATHARTIC VALUE OF THE HISTORY

In addition to its value in diagnosis, history taking is of prognostic and therapeutic value. The mental catharsis, particularly in psychoneurotic disturbances, may result in amelioration of symptoms. This principle is as old as the religious confessional and as new as the Freudian method. A simple type of psychotherapy, it requires no unusual skill or exceptional training. It creates a deep bond of understanding between physician and patient.

CHAPTER 155

PRINCIPLES OF PHYSICAL EXAMINATION

THE physical examination in the office or home imposes upon the examiner a grave responsibility. In institutional work the patient is often seen by admitting physicians, internes, technicians, members of the attending staff and specialists. In private practice, the general practitioner functions as a composite of all branches of medicine; there are no senior officers to furnish additional data or verify findings; technical information can be obtained only at additional trouble and expense.

FUNCTIONS OF THE PHYSICAL EXAMINATION

Each new patient should have a complete physical examination. A complete survey provides a *norm* for each individual and at subsequent visits the subject is reexamined for evidences of deviation from the original findings. Thus it might be established that a given individual in health had a bradycardia or a relative tachycardia; his blood pressure may have been above or below the average levels. This knowledge is of inestimable value in the interpretation of findings encountered at a later date or during illness.

The establishment of the physical status serves as a *health examination*. Irrespective of the presenting symptoms, unsuspected manifestations of chronic disease may be detected before subjective complaints register in the consciousness of the patient. Thus routine palpation of the female breast constitutes one of the most significant practical advances in the early diagnosis of malignancy.

Meticulous physical examination establishes the *doctor-patient relationship*. The patient realizes the broader scope and greater thoroughness of the physician whose interest is manifest in the body as a unity; he appreciates that this type of care is greatly to be cherished; he is encouraged to return to his practitioner for future needs.

The routine survey affords training for the physician in *physical diagnosis*. Thus the attuned ear anticipates a louder transmission of heart sounds in a thin, narrow chest than in one that is heavy and covered by a pendulous breast; sensitized fingertips learn to differentiate tension from true involuntary rigidity (p. 3554). In the same manner the examiner notes the relative frequency with which he encounters such accidental findings as systolic murmurs at the apex or base, a palpable liver edge, a ptosed right kidney, malpositions of the uterus, indurations in the fornices, ragged tonsils, nasal occlusion, deviation of the nasal septum, hemorrhoids and varicosities.

From iterated examination the practitioner develops a critical sense and a balance. He learns to avoid the error of explaining prevailing symptoms necessarily on the basis of the physical findings; he registers the deviations as facts and then considers whether they are relevant to the clinical problem; he learns the wide variety of the normal and the true concept of

relativity he does not confuse the *normal* and its variations with the *average* and its wide range. The integrated practitioner realizes that the weight, height, blood pressure, pulse rate or basal metabolic rate of the individual patient may vary widely from the average; he treats his patient and not the thermometer, sphygmomanometer or electrocardiogram.

The performance of the physical examination has tangible *therapeutic value*. This is illustrated particularly in patients who suffer from an anxiety. The knowledge that thorough investigation has revealed no evidence of organic difficulty serves to allay insecurity and may result in amelioration of symptoms.

RECORD KEEPING

The institutional physician must employ the routine charts that are provided by the medical school or the hospital. In private practice the more experienced doctor discards these formal records; they are rarely spaced correctly and they invite mental inertia and the type of careless work that is characterized by entries of crosses, checks and the obnoxious term "negative." For the bulky hospital format the private practitioner substitutes a hand written or typed transcript of the positive findings.

CHAPTER 156

THE PHYSICAL EXAMINATION STATISTICAL DATA AND GENERAL OBSERVATIONS

THE statistical data include notations of age sex marital state economic status height weight body temperature respiratory and pulse rates and blood pressure readings

AGE

The Young—Congenital abnormalities and alimentary disorders are most commonly seen in the newborn Through increasing contact with the external world the child of pre school age is exposed to infections particularly tuberculosis and the exanthems

Puberty and Adolescence—At puberty and adolescence boys and young men may be exposed to venereal infection girls and young women face the extra hazards of the physiological derangements related to menstruation and pregnancy The emotional disorders of pre adolescence and adolescence are encountered schizophrenia takes its toll of human usefulness

Middle Age—In the third fourth and fifth decades appear the late results of youthful infection manifestations of decrecence the onset of degenerative disease particularly *arteriosclerosis* and the ever increasing shadow of *malignancy*

Senility—In the aged the results of *organ atrophy* and the tissue changes of senescence dominate the clinical scene though *malignant disease* and *vascular afflictions* take a heavy toll

At any time in life accidental hazards are important factors in the production of mortality The peacetime incidence of traffic accidents has assumed almost gargantuan proportions War casualties not only menace the soldier but now offer constant threat to the civilian non-combatant community

SEX

Variations in the Female—*Menstrual disorders* and afflictions incident to *childbearing* and parturition are important factors in the interpretation of clinical symptoms in women The female is more prone to develop *cholelithiasis carcinoma of the breast hyperthyroidism* and *hypertension*

Variations in the Male—In the male there is an increased incidence of *gastroduodenal ulcer angina pectoris* and *coronary thrombosis* In the hospitals for chronic diseases the age of the male population is almost a decade younger than that of the female suggesting the toll of wage earning and economic stress In almost any community widows prevail The male debacle thus occurs earlier and his death is usually premature in relation to that of his spouse

MARITAL STATE

The Female—In the married female *pregnancy* and its constitutional effects are important considerations in any clinical disturbance particu

larly the *acute abdominal syndromes*. Accidents of ovulation and ectopic pregnancy or tubal infection may give rise to clinical symptoms readily confused with other types of intraperitoneal disturbances. The possibility of pregnancy or pelvic inflammatory disease secondary to *gonorrheal infection* or resulting from *illegal abortion* must be considered also in unmarried women. Uncharitable though it may seem when faced with the problem of intra abdominal disease in the female particularly if it is pelvic in situation the practitioner must suspect the probability of some process related to pregnancy abortion or venereal infection.

The Male.—In the male there is very little clinical difference between bachelor and benedict. The unattached male is more likely to develop *venereal disease* and the *digestive distresses* associated with irregular eating habits.

In either sex transmissible disease may affect the marital partner. A history of repeated spontaneous abortion suggests the possibility of *syphilis in the husband*. Tuberculosis of a spouse demands a search for the infection in the other members of the family.

RACE RELIGION AND NATIONALITY

Ethnological influences are occasional factors in the incidence and manifestations of disease. Thus the *Negro* the *Polynesian* the *Oriental* and the *Irish* have a lessened resistance to tuberculosis. The *Jew* is particularly liable to suffer from diabetes mellitus and thromboangitis obliterans.

Gout fibrositis and the various types of arthritis are particularly prevalent among the inhabitants of the *British Isles*; dental decay often occurs early and extensively. *Italians* display an increased incidence of diseases of the liver and spleen; cirrhoses are frequently observed. Banti's syndrome and rare types of anemia associated with hepatosplenomegaly are disproportionately common. The *underprivileged Negro* shows a high incidence of syphilitic infection and is frequently subject to essential hypertension. Diabetes mellitus is somewhat less common than in the white. Hyperthyroidism psychoneuroses and schizophrenia are rare in the full blooded Negro; pernicious anemia is almost never seen whereas sickle cell anemia is a disease peculiar to this group.

Racial disease is often geographic rather than ethnological in origin. Patients of any group who have lived for considerable periods in tropical and semitropical regions may suffer from parasitic diseases such as malaria amebiasis echinococcus infestation or schistosomiasis. The last named disease is common among *Puerto Ricans*. The *Chinese* are prone to infestation with liver and lung fluke. Occasionally racial disease actually depends on cultural habits such as diet. For instance because of their low fat diet the Chinese are singularly free from atherosclerosis and coronary thrombosis.

ECONOMIC FACTORS

In the determination of disease economic considerations are of greater significance than racial and geographic factors. The *underprivileged* are more likely to develop syphilis tuberculosis the acute exanthems rheumatic fever hookworm disease and vitamin deficiencies. Crowding

bad housing and malnutrition are important accessories in the pathogenesis of disease. The *rich* and *overindulgent* are candidates for diabetes mellitus, coronary artery disease, and hypertension.

HEIGHT

Normal Height and Growth—At birth the normal baby measures approximately 20 inches. The child grows 9 inches in the first year of life, the boys averaging fractionally more than the girls. (Tables 164 and 166)

TABLE 164.—WEIGHT—HEIGHT—AGE TABLE FOR BOYS FROM BIRTH TO SCHOOL AGE

[illegible]

Weight must be tested to determine if the weight is added or subtracted from the net weight.

35 to 39 $1\frac{1}{4}$ po hd 40 to 44 in $1\frac{1}{2}$ po d

the National Education Association of the United States.

Complete adult stature is reached between the fourteenth and twentieth years. The rate of growth at first is 4 inches annually. Some children continuing at this rate reach full growth at an early age. Others growing more gradually at a decreasing rate do not attain their full growth until the twentieth year. (Tables 165 and 167)

The height of the normal American male varies between 60 and 74 inches. The average is 68 inches (Table 168). Women vary in height between 58 and 70 inches with an average of 64 inches (Table 169). See the following tables of differential diagnosis: Increased Growth (p 692), Decreased Growth (p 693), Diminution in Adult Size (p 694) and Increased Growth in Infancy (p 2779).

WEIGHT

Normal Weight and Gain in Weight—The weight of the newborn infant averages 7 pounds. Variations within normal limits range from 5 to 10

TABLE 165—W. —H o r—A o T Boy o School A

| Height in | Weight at birth (pounds) | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 |
|--------------|--------------------------------|----|----|----|----|----|----|-----|-----|-----|-----|-----|-----|-----|
| 38 | 54 | 54 | 54 | | | | | | | | | | | |
| 39 | 55 | 55 | 55 | | | | | | | | | | | |
| 40 | 56 | 56 | 56 | | | | | | | | | | | |
| 41 | 58 | 58 | 58 | 58 | | | | | | | | | | |
| 42 | 59 | 59 | 59 | 59 | 59 | | | | | | | | | |
| 43 | 41 | 41 | 41 | 41 | 41 | | | | | | | | | |
| 44 | 43 | 43 | 43 | 43 | 43 | | | | | | | | | |
| 45 | 46 | 46 | 46 | 46 | 46 | 46 | | | | | | | | |
| 46 | 48 | 47 | 48 | 48 | 48 | 48 | | | | | | | | |
| 47 | 50 | 49 | 50 | 50 | 50 | 50 | 50 | | | | | | | |
| 48 | 51 | | 52 | 52 | 52 | 52 | 52 | | | | | | | |
| 49 | 53 | | 53 | 53 | 53 | 53 | 53 | 55 | | | | | | |
| 50 | 58 | | 57 | 58 | 58 | 58 | 58 | 58 | 58 | | | | | |
| 51 | 61 | | | 61 | 61 | 61 | 61 | 61 | 61 | | | | | |
| 52 | 64 | | | 63 | 64 | 64 | 64 | 64 | 64 | 64 | | | | |
| 53 | 68 | | | 68 | 67 | 67 | 67 | 67 | 68 | 68 | | | | |
| 54 | 71 | | | | 70 | 70 | 70 | 70 | 71 | 71 | 72 | | | |
| 55 | 74 | | | | 72 | 72 | 72 | 72 | 74 | 74 | 74 | | | |
| 56 | 78 | | | | 73 | 73 | 73 | 73 | 77 | 77 | 78 | 80 | | |
| 57 | 82 | | | | | 79 | 80 | 81 | 81 | 82 | 83 | 85 | | |
| 58 | 85 | | | | | 85 | 84 | 84 | 85 | 85 | 86 | 87 | | |
| 59 | 89 | | | | | | 87 | 88 | 89 | 89 | 90 | 90 | 90 | |
| 60 | 94 | | | | | | 91 | 92 | 92 | 95 | 94 | 9 | 96 | |
| 61 | 99 | | | | | | | 95 | 96 | 97 | 99 | 101 | 103 | 106 |
| 62 | 104 | | | | | | | 100 | 101 | 102 | 105 | 104 | 107 | 111 |
| 63 | 111 | | | | | | | 105 | 106 | 107 | 108 | 110 | 113 | 118 |
| 64 | 117 | | | | | | | | 109 | 111 | 113 | 115 | 117 | 121 |
| 65 | 123 | | | | | | | | 114 | 117 | 118 | 120 | 122 | 127 |
| 66 | 129 | | | | | | | | | 119 | 122 | 125 | 128 | 132 |
| 67 | 133 | | | | | | | | | 124 | 128 | 130 | 134 | 136 |
| 68 | 139 | | | | | | | | | | 134 | 134 | 137 | 141 |
| 69 | 144 | | | | | | | | | | 137 | 139 | 143 | 146 |
| 70 | 147 | | | | | | | | | | 143 | 144 | 145 | 148 |
| 71 | 152 | | | | | | | | | | 148 | 151 | 151 | 152 |
| 72 | 157 | | | | | | | | | | | 153 | 155 | 159 |
| 73 | 163 | | | | | | | | | | | 157 | 160 | 162 |
| 74 | 169 | | | | | | | | | | | 160 | 164 | 168 |

| AGE—YEARS | | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 |
|-----------|---------|----|----|----|----|----|----|----|----|----|----|----|----|
| A | g t ght | 43 | 45 | 47 | 49 | 51 | 53 | 54 | 56 | 58 | 60 | 62 | 64 |
| | Med m | 46 | 48 | 50 | 52 | 54 | 56 | 58 | 60 | 63 | 65 | 67 | 68 |
| | T ll | 49 | 51 | 53 | 55 | 57 | 59 | 61 | 64 | 67 | 70 | 72 | 72 |
| A | g | 5 | 4 | 5 | 5 | 5 | 4 | 8 | 9 | 11 | 14 | 15 | 7 |
| | Med m | 4 | 5 | 6 | 6 | 6 | 7 | 9 | 11 | 15 | 11 | 8 | 4 |
| | T ll | 5 | 7 | 7 | 7 | 7 | 8 | 12 | 15 | 11 | 9 | 7 | 3 |

I der t t nd th g f tables so to l d w ghts f hldren w t ll h rter th those
th se gr p th h bee d d d atarr d ght t m t d w ght All th th w ghts rep se t gea f
h p b h ght d g bseved th t dy
P e p ed by B d T B l d w Ph D I w Chld W l f T se h Stat St t U rsty f I w d
Th mas D Wood M D Col mb U ty N Y C Repr: t i by pe so f th Am r. Cl l f W l f re
A soci t d th N t l Ed catio A soci t f th U t d stat

pounds After an initial loss of 6 to 10 ounces preventable by judicious feeding the weight curve turns upward. It doubles between the fourth and sixth month and usually trebles by the end of the year (Tables 164 and 166)

From the first to the twelfth year the normal child makes an average gain of 4 or 5 pounds per year the girls weighing slightly less than the boys

At the menarche which commonly occurs between the tenth and fourteenth years the weight of the female increases so rapidly that she out scales the average male With the advent of male secondary sex characteristics in the eleventh to the fifteenth year a sufficiently greater increase in growth and weight occurs so that the average boy is again taller and heavier than the average girl (Tables 165 and 167) This ratio prevails through adult life

TABLE 168—WEIGHT—HEIGHT—AGE TABLE FOR GIRLS FROM BIRTH TO SCHOOL AGE

| Height in feet | Average Weight in pounds | 1 yr | 3 yr | 6 mo | 9 mo | 12 mo | 18 mo | 24 mo | 30 mo | 36 mo | 48 mo | 60 mo | 72 mo |
|-------------------|--------------------------------|---------|---------|---------|---------|----------|----------|----------|----------|----------|----------|----------|----------|
| 20 | 8 | 8 | | | | | | | | | | | |
| 21 | 9 | 9 | 10 | | | | | | | | | | |
| 22 | 10½ | 10 | 11 | | | | | | | | | | |
| 23 | 12 | 11 | 12 | 13 | | | | | | | | | |
| 24 | 13½ | 12 | 13 | 14 | 14 | | | | | | | | |
| 25 | 15 | 13 | 14 | 15 | 15 | | | | | | | | |
| 26 | 16½ | | 15 | 16 | 17 | 17 | | | | | | | |
| 27 | 17½ | | 16 | 17 | 18 | 18 | | | | | | | |
| 28 | 19 | | | 17 | 18 | 19 | 19 | | | | | | |
| 29 | 20 | | | 19 | 20 | 20 | 20 | | | | | | |
| 30 | 21½ | | | 21 | 21 | 21 | 21 | 21 | | | | | |
| 31 | 23½ | | | | 22 | 22 | 23 | 23 | 23 | | | | |
| 32 | 24 | | | | | 23 | 24 | 24 | 24 | 25 | | | |
| 33 | 25 | | | | | | 25 | 25 | 25 | 26 | | | |
| 34 | 26½ | | | | | | 26 | 26 | 26 | 27 | | | |
| 35 | 29 | | | | | | 29 | 29 | 29 | 29 | 29 | | |
| 36 | 30 | | | | | | | 30 | 30 | 30 | 30 | 31 | |
| 37 | 31½ | | | | | | | 31 | 31 | 31 | 31 | 32 | |
| 38 | 33½ | | | | | | | 31 | 32 | 33 | 33 | 34 | |
| 39 | 34 | | | | | | | 31 | 31 | 31 | 31 | 34 | 34 |
| 40 | 35½ | | | | | | | | | | | | |
| 41 | 37½ | | | | | | | | | 35 | 36 | 36 | 36 |
| 42 | 39 | | | | | | | | | | 37 | 37 | 37 |
| 43 | 41 | | | | | | | | | | 39 | 39 | 39 |
| 44 | 42½ | | | | | | | | | | 40 | 41 | 41 |
| | | | | | | | | | | | | 42 | 42 |
| 5 | 45 | | | | | | | | | | | | 45 |
| 46 | 47½ | | | | | | | | | | | | 47 |
| 47 | 50 | | | | | | | | | | | | 50 |
| 48 | 52½ | | | | | | | | | | | | 52 |

Weight in pounds, height in feet and inches, age in years, months, and days. The average weight and height of the child are given in the first column. The average weight and height of the child are given in the first column.

35 to 39 1 pound 45 to 49 1½ pounds 40 to 44 1½ pounds

Prepared by Robert M. Woodbury, Ph.D., reported by permission of the American Child Health Association, National Educational Association of the United States.

The standard weights of the average adult American male and female vary according to age and height (Tables 168 and 169) These height weight and age scales are based on averages

Deviations in Weight—Normal weight may deviate so widely from the average that it is not a simple matter to decide whether a given individual is above or below optimum In a general way a variation from the standard weights of the insurance tables of more than twenty pounds may be taken as exceeding the limits of normality A male five feet eight inches tall at age thirty should weigh 152 pounds A weight of 130 pounds may be considered as slightly less and of 175 pounds slightly more than normal

TABLE 167—W T—H T—AG TABLE FOR GIRLS OF SCHOOL AGE

| Height inches | Age Weight inches (inches) | 5 y age | 6 y age | 7 y | 8 y | 9 y | 10 y | 11 y | 12 y | 13 y | 14 y | 15 y | 16 y | 17 y |
|------------------|-------------------------------------|---------|---------|-----|-----|-----|------|------|------|------|------|------|------|------|
| 38 | 33 | 33 | 33 | | | | | | | | | | | |
| 39 | 34 | 34 | 34 | | | | | | | | | | | |
| 40 | 35 | 35 | 35 | 35 | | | | | | | | | | |
| 41 | 36 | 36 | 36 | 36 | | | | | | | | | | |
| 42 | 37 | 37 | 37 | 37 | | | | | | | | | | |
| 43 | 38 | 38 | 38 | 38 | | | | | | | | | | |
| 44 | 39 | 39 | 39 | 39 | | | | | | | | | | |
| 45 | 40 | 40 | 40 | 40 | 41 | | | | | | | | | |
| 46 | 41 | 41 | 41 | 41 | 41 | | | | | | | | | |
| 47 | 42 | 42 | 42 | 42 | 42 | | | | | | | | | |
| 48 | 43 | 43 | 43 | 43 | 43 | | | | | | | | | |
| 49 | 44 | 44 | 44 | 44 | 44 | | | | | | | | | |
| 50 | 45 | 45 | 45 | 45 | 45 | | | | | | | | | |
| 51 | 46 | 46 | 46 | 46 | 46 | | | | | | | | | |
| 52 | 47 | 47 | 47 | 47 | 47 | | | | | | | | | |
| 53 | 48 | 48 | 48 | 48 | 48 | | | | | | | | | |
| 54 | 49 | 49 | 49 | 49 | 49 | | | | | | | | | |
| 55 | 50 | 50 | 50 | 50 | 50 | | | | | | | | | |
| 56 | 51 | 51 | 51 | 51 | 51 | | | | | | | | | |
| 57 | 52 | 52 | 52 | 52 | 52 | | | | | | | | | |
| 58 | 53 | 53 | 53 | 53 | 53 | | | | | | | | | |
| 59 | 54 | 54 | 54 | 54 | 54 | | | | | | | | | |
| 60 | 55 | 55 | 55 | 55 | 55 | | | | | | | | | |
| 61 | 56 | 56 | 56 | 56 | 56 | | | | | | | | | |
| 62 | 57 | 57 | 57 | 57 | 57 | | | | | | | | | |
| 63 | 58 | 58 | 58 | 58 | 58 | | | | | | | | | |
| 64 | 59 | 59 | 59 | 59 | 59 | | | | | | | | | |
| 65 | 60 | 60 | 60 | 60 | 60 | | | | | | | | | |
| 66 | 61 | 61 | 61 | 61 | 61 | | | | | | | | | |
| 67 | 62 | 62 | 62 | 62 | 62 | | | | | | | | | |
| 68 | 63 | 63 | 63 | 63 | 63 | | | | | | | | | |
| 69 | 64 | 64 | 64 | 64 | 64 | | | | | | | | | |
| 70 | 65 | 65 | 65 | 65 | 65 | | | | | | | | | |
| 71 | 66 | 66 | 66 | 66 | 66 | | | | | | | | | |
| 72 | 67 | 67 | 67 | 67 | 67 | | | | | | | | | |
| 73 | 68 | 68 | 68 | 68 | 68 | | | | | | | | | |
| 74 | 69 | 69 | 69 | 69 | 69 | | | | | | | | | |
| 75 | 70 | 70 | 70 | 70 | 70 | | | | | | | | | |
| 76 | 71 | 71 | 71 | 71 | 71 | | | | | | | | | |
| 77 | 72 | 72 | 72 | 72 | 72 | | | | | | | | | |
| 78 | 73 | 73 | 73 | 73 | 73 | | | | | | | | | |
| 79 | 74 | 74 | 74 | 74 | 74 | | | | | | | | | |
| 80 | 75 | 75 | 75 | 75 | 75 | | | | | | | | | |
| 81 | 76 | 76 | 76 | 76 | 76 | | | | | | | | | |
| 82 | 77 | 77 | 77 | 77 | 77 | | | | | | | | | |
| 83 | 78 | 78 | 78 | 78 | 78 | | | | | | | | | |
| 84 | 79 | 79 | 79 | 79 | 79 | | | | | | | | | |
| 85 | 80 | 80 | 80 | 80 | 80 | | | | | | | | | |
| 86 | 81 | 81 | 81 | 81 | 81 | | | | | | | | | |
| 87 | 82 | 82 | 82 | 82 | 82 | | | | | | | | | |
| 88 | 83 | 83 | 83 | 83 | 83 | | | | | | | | | |
| 89 | 84 | 84 | 84 | 84 | 84 | | | | | | | | | |
| 90 | 85 | 85 | 85 | 85 | 85 | | | | | | | | | |
| 91 | 86 | 86 | 86 | 86 | 86 | | | | | | | | | |
| 92 | 87 | 87 | 87 | 87 | 87 | | | | | | | | | |
| 93 | 88 | 88 | 88 | 88 | 88 | | | | | | | | | |
| 94 | 89 | 89 | 89 | 89 | 89 | | | | | | | | | |
| 95 | 90 | 90 | 90 | 90 | 90 | | | | | | | | | |
| 96 | 91 | 91 | 91 | 91 | 91 | | | | | | | | | |
| 97 | 92 | 92 | 92 | 92 | 92 | | | | | | | | | |
| 98 | 93 | 93 | 93 | 93 | 93 | | | | | | | | | |
| 99 | 94 | 94 | 94 | 94 | 94 | | | | | | | | | |
| 100 | 95 | 95 | 95 | 95 | 95 | | | | | | | | | |
| 101 | 96 | 96 | 96 | 96 | 96 | | | | | | | | | |
| 102 | 97 | 97 | 97 | 97 | 97 | | | | | | | | | |
| 103 | 98 | 98 | 98 | 98 | 98 | | | | | | | | | |
| 104 | 99 | 99 | 99 | 99 | 99 | | | | | | | | | |
| 105 | 100 | 100 | 100 | 100 | 100 | | | | | | | | | |
| 106 | 101 | 101 | 101 | 101 | 101 | | | | | | | | | |
| 107 | 102 | 102 | 102 | 102 | 102 | | | | | | | | | |
| 108 | 103 | 103 | 103 | 103 | 103 | | | | | | | | | |
| 109 | 104 | 104 | 104 | 104 | 104 | | | | | | | | | |
| 110 | 105 | 105 | 105 | 105 | 105 | | | | | | | | | |
| 111 | 106 | 106 | 106 | 106 | 106 | | | | | | | | | |
| 112 | 107 | 107 | 107 | 107 | 107 | | | | | | | | | |
| 113 | 108 | 108 | 108 | 108 | 108 | | | | | | | | | |
| 114 | 109 | 109 | 109 | 109 | 109 | | | | | | | | | |
| 115 | 110 | 110 | 110 | 110 | 110 | | | | | | | | | |
| 116 | 111 | 111 | 111 | 111 | 111 | | | | | | | | | |
| 117 | 112 | 112 | 112 | 112 | 112 | | | | | | | | | |
| 118 | 113 | 113 | 113 | 113 | 113 | | | | | | | | | |
| 119 | 114 | 114 | 114 | 114 | 114 | | | | | | | | | |
| 120 | 115 | 115 | 115 | 115 | 115 | | | | | | | | | |
| 121 | 116 | 116 | 116 | 116 | 116 | | | | | | | | | |
| 122 | 117 | 117 | 117 | 117 | 117 | | | | | | | | | |
| 123 | 118 | 118 | 118 | 118 | 118 | | | | | | | | | |
| 124 | 119 | 119 | 119 | 119 | 119 | | | | | | | | | |
| 125 | 120 | 120 | 120 | 120 | 120 | | | | | | | | | |
| 126 | 121 | 121 | 121 | 121 | 121 | | | | | | | | | |
| 127 | 122 | 122 | 122 | 122 | 122 | | | | | | | | | |
| 128 | 123 | 123 | 123 | 123 | 123 | | | | | | | | | |
| 129 | 124 | 124 | 124 | 124 | 124 | | | | | | | | | |
| 130 | 125 | 125 | 125 | 125 | 125 | | | | | | | | | |
| 131 | 126 | 126 | 126 | 126 | 126 | | | | | | | | | |
| 132 | 127 | 127 | 127 | 127 | 127 | | | | | | | | | |
| 133 | 128 | 128 | 128 | 128 | 128 | | | | | | | | | |
| 134 | 129 | 129 | 129 | 129 | 129 | | | | | | | | | |
| 135 | 130 | 130 | 130 | 130 | 130 | | | | | | | | | |
| 136 | 131 | 131 | 131 | 131 | 131 | | | | | | | | | |
| 137 | 132 | 132 | 132 | 132 | 132 | | | | | | | | | |
| 138 | 133 | 133 | 133 | 133 | 133 | | | | | | | | | |
| 139 | 134 | 134 | 134 | 134 | 134 | | | | | | | | | |
| 140 | 135 | 135 | 135 | 135 | 135 | | | | | | | | | |
| 141 | 136 | 136 | 136 | 136 | 136 | | | | | | | | | |
| 142 | 137 | 137 | 137 | 137 | 137 | | | | | | | | | |
| 143 | 138 | 138 | 138 | 138 | 138 | | | | | | | | | |
| 144 | 139 | 139 | 139 | 139 | 139 | | | | | | | | | |
| 145 | 140 | 140 | 140 | 140 | 140 | | | | | | | | | |
| 146 | 141 | 141 | 141 | 141 | 141 | | | | | | | | | |
| 147 | 142 | 142 | 142 | 142 | 142 | | | | | | | | | |
| 148 | 143 | 143 | 143 | 143 | 143 | | | | | | | | | |
| 149 | 144 | 144 | 144 | 144 | 144 | | | | | | | | | |
| 150 | 145 | 145 | 145 | 145 | 145 | | | | | | | | | |
| 151 | 146 | 146 | 146 | 146 | 146 | | | | | | | | | |
| 152 | 147 | 147 | 147 | 147 | 147 | | | | | | | | | |
| 153 | 148 | 148 | 148 | 148 | 148 | | | | | | | | | |
| 154 | 149 | 149 | 149 | 149 | 149 | | | | | | | | | |
| 155 | 150 | 150 | 150 | 150 | 150 | | | | | | | | | |
| 156 | 151 | 151 | 151 | 151 | 151 | | | | | | | | | |
| 157 | 152 | 152 | 152 | 152 | 152 | | | | | | | | | |
| 158 | 153 | 153 | 153 | 153 | 153 | | | | | | | | | |
| 159 | 154 | 154 | 154 | 154 | 154 | | | | | | | | | |
| 160 | 155 | 155 | 155 | 155 | 155 | | | | | | | | | |
| 161 | 156 | 156 | 156 | 156 | 156 | | | | | | | | | |
| 162 | 157 | 157 | 157 | 157 | 157 | | | | | | | | | |
| 163 | 158 | 158 | 158 | 158 | 158 | | | | | | | | | |
| 164 | 159 | 159 | 159 | 159 | 159 | | | | | | | | | |
| 165 | 160 | 160 | 160 | 160 | 160 | | | | | | | | | |
| 166 | 161 | 161 | 161 | 161 | 161 | | | | | | | | | |
| 167 | 162 | 162 | 162 | 162 | 162 | | | | | | | | | |
| 168 | 163 | 163 | 163 | 163 | 163 | | | | | | | | | |
| 169 | 164 | 164 | 164 | 164 | 164 | | | | | | | | | |
| 170 | 165 | 165 | 165 | 165 | 165 | | | | | | | | | |
| 171 | 166 | 166 | 166 | 166 | 166 | | | | | | | | | |
| 172 | 167 | 167 | 167 | 167 | 167 | | | | | | | | | |
| 173 | 168 | 168 | 168 | 168 | 168 | | | | | | | | | |
| 174 | 169 | 169 | 169 | 169 | 169 | | | | | | | | | |
| 175 | 170 | 170 | 170 | 170 | 170 | | | | | | | | | |
| 176 | 171 | 171 | 171 | 171 | 171 | | | | | | | | | |
| 177 | 172 | 172 | 172 | 172 | 172 | | | | | | | | | |
| 178 | 173 | 173 | 173 | 173 | 173 | | | | | | | | | |

Clinically it is of more importance to note an *excessive gain or excessive loss of weight*. Wholly aside from the cause of weight loss and the diagnostic problems related thereto the excessively thin individual tends to be low in stamina diminished in resistance and prone to infection. The excessively stocky individual is a more likely candidate for the metabolic diseases particularly diabetes mellitus angina pectoris coronary occlusion and arteriosclerosis. See *Differential Diagnosis of Gain in Weight* (p 695) *Loss of Weight* (p 700) *Loss of Weight in Infancy* (p 2784)

BODY TEMPERATURE

Use of the Clinical Thermometer—The body temperature may be taken orally in the axilla or by rectum. The *rectal temperature* alone is accurate and reliable. Axillary and oral temperatures are subject to many variations due to extraneous causes. In children particularly a misleading rectal reading may be recorded after exercise or following an evacuation an enema

TABLE 169—HEIGHT—WEIGHT—AGE—RELATIONS TABLE OF AVERAGE HEIGHTS AND WEIGHTS—WOMEN

| | 15 y s | 20 yr | 25 yr | 30 y | 35 yrs. | 40 y s. | 45 yrs. | 50 y s. | 55 yrs. |
|------------|--------|-------|-------|------|---------|---------|---------|---------|---------|
| 4 ft 8 in | 101 | 106 | 109 | 112 | 115 | 119 | 122 | 125 | 126 |
| 4 ft 9 in | 103 | 108 | 111 | 114 | 117 | 121 | 124 | 127 | 127 |
| 4 ft 10 in | 105 | 110 | 113 | 116 | 119 | 123 | 126 | 129 | 129 |
| 4 ft 11 in | 106 | 112 | 115 | 118 | 121 | 125 | 129 | 131 | 131 |
| 5 ft 0 in | 107 | 114 | 117 | 120 | 123 | 127 | 130 | 133 | 133 |
| 5 ft 1 in | 109 | 116 | 119 | 122 | 125 | 129 | 132 | 135 | 135 |
| 5 ft 2 in | 112 | 119 | 121 | 124 | 127 | 132 | 135 | 138 | 138 |
| 5 ft 3 in | 113 | 122 | 124 | 127 | 130 | 135 | 139 | 141 | 141 |
| 5 ft 4 in | 118 | 123 | 128 | 131 | 134 | 138 | 141 | 144 | 144 |
| 5 ft 5 in | 122 | 128 | 131 | 134 | 138 | 142 | 145 | 148 | 148 |
| 5 ft 6 in | 126 | 132 | 135 | 138 | 142 | 146 | 149 | 152 | 153 |
| 5 ft 7 in | 130 | 136 | 139 | 142 | 146 | 150 | 153 | 156 | 158 |
| 5 ft 8 in | 134 | 140 | 143 | 146 | 150 | 154 | 157 | 161 | 163 |
| 5 ft 9 in | 138 | 145 | 147 | 150 | 154 | 158 | 161 | 165 | 167 |
| 5 ft 10 in | 142 | 147 | 151 | 154 | 157 | 161 | 164 | 169 | 171 |
| 5 ft 11 in | 147 | 151 | 154 | 157 | 160 | 164 | 169 | 173 | 174 |
| 6 ft 0 in | 152 | 156 | 158 | 161 | 165 | 167 | 171 | 176 | 177 |

From L. F. E. on I. t. t. e.

or a hot bath. In collapse the oral temperature may be normal or subnormal whereas the rectal reading is elevated.

The Range of Normal Rectal Temperature—The registration of the clinical thermometer is subject to interpretation. It is a common belief among the laity that any reading other than that on the arrow at 98.6° F represents a deviation from normal. The patient may thus report that he "has fever" meaning that the thermometer reads above the arrow.

The average rectal temperature is commonly believed to be from 99° to 99.6° F. Many individuals however normally run a subaverage temperature as low as 97.6° others record a reading above average which may in health approach 100°.

Daily Fluctuations in Normal Temperature—Besides the normal variations in different individuals there is a daily fluctuation of approximately one degree in any given individual. The highest reading is commonly obtained at 4 P.M. the lowest at 4 A.M. In night workers the reverse may be true.

When the practitioner obtains a single reading in the office or during the house visit the presence of low grade or relative pyrexia particularly

as seen in incipient tuberculosis may be overlooked. For example in a patient whose normal reading is set between 98 and 99 a morning reading at 99.6° indicates fever. A similar reading occurring in an individual whose span is between 99 and 100 may have little or no significance particularly if it is the afternoon reading.

Absolute and Relative Pyrexia—Pyrexia may be absolute or relative. A rectal temperature in excess of 100° is *absolute pyrexia*. Occasionally the physician encounters the rare individual whose normal body temperature in health may slightly exceed 100 (sympathetic fever).

Relative pyrexia may be present in individuals whose highest recorded temperature is less than 100. This may be recognized by recording the body temperature at intervals of four hours. The fluctuation should not exceed one degree. For example in incipient tuberculosis a variation between 98 and 99.6° (span of 1.6°) constitutes a relative pyrexia and suggests the presence of active infection.

The variations and the methods of registering bodily temperatures are stressed because of the carelessness of the usual observations.

See *Differential Diagnosis of Fever* (p 19) *Septic Pyrexia* (p 23) *Fever in Pregnancy* (p 264°) *Chills* (p 31) *Cryptogenic Fever* (p 26) *Fever in Infancy* (p 2760) *Fever in Aged* (p 980) *Eruptive Fevers* (p 172) *Infectious Fevers* (p 30) *Post operative Fever* (p 4007) *Metabolic Fevers* (p 718) *Intrathoracic Fever* (p 404) *Acute Hypopyrexia* (p 22) and *Sustained Hypopyrexia* (p 21)

THE RESPIRATORY RATE

The Normal Rate and Rhythm—The normal adult individual breathes fourteen to twenty times per minute. The respiratory rate is more rapid in the newborn in infancy and childhood. It is slower during sleep and in the aged. Irregularities in depth and rate occur normally during sleep particularly in the young.

See *Differential Diagnosis of Bradypnea Oligopnea and Apnea* (p 2014) *Hyperpnea Tachypnea Polypnea Dyspnea Orthopnea* and *Respiratory Irregularities* (p 2016)

THE PULSE RATE

The Normal and Its Variations—The normal pulse rate is commonly stated to be 72 beats to the minute. This however is an average rather than a normal figure. Certain individuals in health notably athletes in training may have a rate as low as 44 as the result of excessive vagal activity. Other individuals particularly women and children are adrenergic and may normally record a rate approaching 100.

The terms *bradycardia* and *tachycardia* are therefore distinctly relative. Thus a rate of 60 is relative tachycardia for an individual whose normal count is in the 40's. A similar rate would be relative bradycardia for the adrenergic patient whose normal rate is set in the upper 80's.

The practitioner must learn the normal rate for each individual patient. The pulse rate should be recorded under relatively basal conditions that is when the temperature is normal and the individual is in a post absorptive state mentally and physically at rest.

See *Differential Diagnosis of Bradycardia* (p 875) and *Tachycardia* (p 877)

Clinically it is of more importance to note an *excessive gain* or *excessive loss* of weight. Wholly aside from the cause of weight loss and the diagnostic problems related thereto the *excessively thin individual* tends to be low in stamina diminished in resistance and prone to infection. The *excessively stocky individual* is a more likely candidate for the metabolic diseases particularly diabetes mellitus angina pectoris, coronary occlusion and arteriosclerosis. See *Differential Diagnosis of Gain in Weight* (p 695) *Loss of Weight* (p 700) *Loss of Weight in Infancy* (p 2784)

BODY TEMPERATURE

Use of the Clinical Thermometer—The body temperature may be taken orally in the axilla or by rectum. The *rectal temperature* alone is accurate and reliable. Axillary and oral temperatures are subject to many variations due to extraneous causes. In children particularly a misleading rectal reading may be recorded after exercise or following an evacuation an enema

I E 169—HEIGHT—WEIGHT—AGE—RELATIO TABLE OF AVE OF HEIGHTS AND WEIGHTS—WOMEN

| | 15 y | 20 y s | 25 yr | 30 y | 35 y | 40 yrs | 45 yrs | 50 y s. | 55 |
|------------|------|--------|-------|------|------|--------|--------|---------|-----|
| 4 ft 8 | 101 | 106 | 109 | 112 | 115 | 119 | 122 | 125 | 128 |
| 4 ft 9 | 103 | 109 | 111 | 114 | 117 | 121 | 124 | 127 | 131 |
| 4 ft 10 | 105 | 110 | 115 | 118 | 119 | 123 | 126 | 129 | 129 |
| 4 ft 11 in | 106 | 112 | 115 | 118 | 121 | 125 | 128 | 131 | 131 |
| 5 ft 0 in | 107 | 114 | 117 | 120 | 123 | 127 | 130 | 133 | 133 |
| 5 ft 1 | 109 | 116 | 119 | 122 | 125 | 129 | 132 | 135 | 135 |
| 5 ft 2 | 112 | 119 | 121 | 124 | 127 | 132 | 135 | 139 | 139 |
| 5 ft 3 | 113 | 122 | 124 | 127 | 130 | 135 | 138 | 141 | 141 |
| 5 ft 4 | 118 | 125 | 128 | 131 | 134 | 138 | 141 | 144 | 144 |
| 5 ft 5 | 122 | 128 | 131 | 134 | 138 | 142 | 145 | 149 | 149 |
| 5 ft 6 | 126 | 132 | 135 | 139 | 142 | 146 | 149 | 152 | 153 |
| 5 ft 7 | 130 | 136 | 139 | 142 | 146 | 150 | 153 | 156 | 158 |
| 5 ft 8 | 134 | 140 | 143 | 146 | 150 | 154 | 157 | 161 | 161 |
| 5 ft 9 | 138 | 144 | 147 | 150 | 154 | 159 | 161 | 165 | 165 |
| 5 ft 10 | 142 | 147 | 151 | 154 | 157 | 161 | 164 | 169 | 171 |
| 5 ft 11 | 147 | 151 | 154 | 157 | 160 | 164 | 168 | 173 | 174 |
| 6 ft 0 | 152 | 156 | 158 | 161 | 165 | 167 | 171 | 176 | 177 |

From Life Expectancy

or a hot bath. In collapse the oral temperature may be normal or subnormal whereas the rectal reading is elevated.

The Range of Normal Rectal Temperature—The registration of the clinical thermometer is subject to interpretation. It is a common belief among the laity that any reading other than that on the arrow at 98.6° F represents a deviation from normal. The patient may thus report that he has fever meaning that the thermometer reads above the arrow.

The average rectal temperature is commonly believed to be from 99° to 99.6° F. Many individuals however normally run a subaverage temperature as low as 97.6° others record a reading above average which may in health approach 100°.

Daily Fluctuations in Normal Temperature—Besides the normal variations in different individuals there is a daily fluctuation of approximately one degree in any given individual. The highest reading is commonly obtained at 4 P.M. the lowest at 4 A.M. In night workers the reverse may be true.

When the practitioner obtains a single reading in the office or during the house visit the presence of low grade or relative pyrexia particularly

Diastolic Pressure—The diastolic pressure can only be estimated by auscultation. After inflation of the cuff for a determination of the systolic pressure the air in the system is gradually permitted to escape. A variety of alterations in quality and intensity of sound are noted. At a certain level more or less abruptly they become muffled. Shortly thereafter they disappear entirely. These varying changes have been divided into as many as five phases but no useful purpose seems to accrue from the differentiation. The muffled phase that precedes the complete cessation of sound is the best approximation of diastolic pressure. Having determined this during deflation a check reading may be made on inflation of the cuff.

Pulse Pressure—The pulse pressure is calculated by subtracting the diastolic from the systolic reading.

Variations in Normal Blood Pressure—Blood pressure readings are subject to wide variation. It is commonly stated that the normal blood pressure of the adult male approximates 120 systolic and 80 diastolic with a pulse pressure of 40. Readings are lower in children and young adults and usually increase progressively with age.

One of the dictums most widely quoted is that the systolic pressure should register 100 plus the age of the patient. Again it is commonly stated that the ratio of systolic to diastolic blood pressure should be as 3 is to 2 of the diastolic blood pressure to pulse pressure as 2 is to 1. These generalities should not be rigidly accepted. Individual blood pressure readings vary as do measurements of height and weight.

Range of Normal Systolic Blood Pressure—The range of the normal systolic pressure in the healthy human may be exceedingly wide. There are any number of normal patients whose systolic blood pressure does not exceed 100 mm of mercury. Again there are many patients particularly those who are highstrung and obese whose normal reading may exceed 150. While the readings in the low normal range have no particularly ominous connotation those in the higher range should be dealt with judiciously. This is particularly true where the higher figure is repeatedly obtained so that it is obviously not the result of the excitement and strangeness of the first visit or a first reading.

In his routine physical examination the practitioner can perform no more important function than to learn the normal blood pressure of his particular patient. Thereafter a progressive fall in blood pressure and more particularly a progressive elevation have great significance. Thus a systolic blood pressure reading of 130 might not have any great importance at a first examination. However if it were known that the reading of the previous year approximated 100 that fact would then become significant.

Range of Normal Diastolic Pressure—It is commonly taught that the diastolic pressure is constant and that significant variations rarely occur. It is believed that the elevation of the diastolic pressure beyond 100 mm of mercury is of evil prognostic significance. Diastolic blood pressure does vary however as does the systolic but on a percentile basis. If the systolic pressure rises from 120 to 200 (an increase of 33 per cent) the diastolic pressure if set at 100 will rise to approximately 133.

Range of Normal Pulse Pressure—The pulse pressure should occupy a band between 25 per cent at the least and 40 per cent at the most.

Pulse Rate in Relation to Basal Metabolic Rate —If the basal pulse rate is recorded as carefully and as accurately as the basal metabolic rate it may prove of equally great significance at much less cost. Once a relationship is established between the pulse rate and the basal metabolic rate the former accurately parallels the latter. Thus in hyperthyroidism with a pulse rate of 130 and a basal metabolic rate of plus 40 per cent a fall of the former to 100 would probably indicate a proportionate fall in the latter. In the hypothyroid patient receiving substitution therapy with a pulse rate of 60 and a basal metabolic rate of minus 20 per cent a rise of the former to 70 would indicate an approach of the latter to normality.

BLOOD PRESSURE

The Sphygmomanometer and Its Use —In the human subject blood pressure readings are recorded by the sphygmomanometer. Such readings should be taken under relatively basal conditions because of the profound effects of physical and emotional factors. The ease with which blood pressure estimations may be made and the universal use of the sphygmomanometer have resulted in a grievous degree of casualness and carelessness in the performance of this exceedingly valuable clinical procedure. At times technical errors may be unavoidable, as in determinations taken in the case of patients with large arms whose readings may be as much as 30 mm. higher than the true intra arterial tension.

Readings taken when the patient is recumbent should be 10 mm. of mercury higher than those obtained in the sitting or upright positions. The horizontal and vertical blood pressure should be obtained in all cases of dizziness and syncope since there may be postural or orthostatic hypotension.

It is important to take the blood pressure in both arms. Differences of 5 to 10 mm. of mercury in the systolic and diastolic pressures are usually encountered. Greater differences are occasionally observed and are of important diagnostic significance (unilateral low blood pressure in aortic aneurysm, scalenus anticus syndrome, patent ductus arteriosus and coarctation of the aorta). Brachial pressure should be checked with the popliteal pressure when coarctation of the aorta is suspected.

Systolic Pressure —With the patient at rest the arm is bared, the compression bag placed over the brachial artery and the cuff applied snugly. The bell of the stethoscope is placed over the brachial artery in the antecubital fossa. By inflating the bulb the pressure in the bag is raised until the arterial sounds are no longer heard. This level is noted. The cuff is then deflated slowly. The pressure is permitted to fall until the sounds are again heard. Commonly the levels of the disappearance of the sounds during inflation and their reappearance during deflation are identical. This reading may be taken as the systolic pressure.

The auscultatory reading may be checked by palpation. The stethoscope is removed. With the fingers of one hand the radial pulse is felt. The bag is again inflated and the level is noted at which the radial pulse can no longer be palpated. The cuff is then deflated and the level is noted at which the pulse rate is again palpable. The two palpatory readings should be identical. They are commonly 10 mm. of mercury less than the auscultatory readings.

be present. The neck is long and slender, the thoracic cage long and narrow. The intercostal angle is acute. The free ribs all but impinge on the pelvic crest.

Visceroptosis (Fig 1011) is the rule, the stomach being of the fish hook type, the transverse colon generally hangs down suspended like a ham mock between the splenic and hepatic flexures. The heart is usually hypoplastic, the blood pressure low, the kidneys, particularly the right, ptosed. In the male, the testes hang low, there is often a varicocele, hernias are

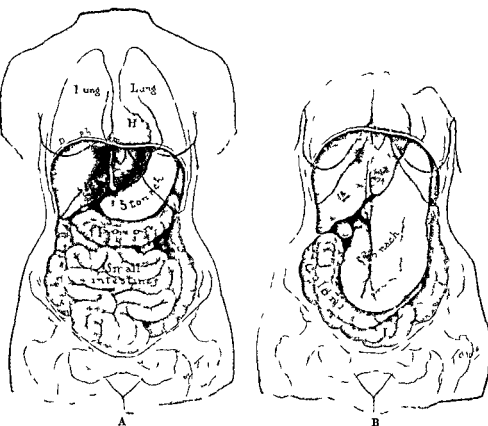


Fig 1011.—Position and relationship of the viscera in the female. A Normal female figure. B Visceroptosis (abdominal ptosis). Gl. nards disease, enteroptosis. position of colon lying behind the stomach indicated by dotted line.

common. These patients commonly have *sway back* with an increase in the lumbar lordosis as well as a dorsal kyphosis. The arches often are flat. Orthostatic albuminuria may occur.

Visceroptotic patients often have marked *vasomotor instability* (p 1395) with cold clammy hands which they interpret as indicative of poor circulation. The complexion is sallow. There are rings under the eyes. The appearance suggests an anemia, but the blood count may be surprisingly

relation to the systolic pressure That is to say a pulse pressure in excess of 40 per cent of the systolic pressure is abnormally high If it is less than 25 per cent of the systolic pressure it is abnormally low

See *Differential Diagnosis of Systolic Hypertension* (p 910) *Hypotension* (p 917) *Blood Pressure Anomalies Other than Those Related to Systolic Tension* (p 918)

GENERAL OBSERVATIONS

After the collection of statistical data many general observations are recorded Without formality the practitioner notes the general state of

TABLE 170—GENERAL OBSERVATIONS

| | Descriptive Remarks | Diagnosis Reference |
|-------------------------------|--|---------------------|
| State of Health | Apparently well acutely ill prostrated in acute distress dyspneic coughing in pain | |
| Nutrition | Well nourished poorly nourished cachectic obese emaciated | (p 69) |
| State of Development | Flabby muscular asymmetrical | (p 299) |
| Secondary Sex Characteristics | Consistent with age infantile masculinization in female feminization in male | (p 2480) |
| Mentality | Normal precocious retarded | (p 1333) |
| Consciousness | Alert clouded comatose confused | (p 1001) |
| Attention | Normal distractable blunted | (p 106) |
| Thought | Clear delusional hypochondriacal blocked obsessive retarded manic | (p 129) |
| Orientation | Consistent with educational and social opportunities defective | (p 1295) |
| Judgment | Clear lacking in insight | (p 1295) |
| Memory | For recent past (menu of last meal) for remote past amnesia falsification | (p 1296) |
| Affect | Agitated anxious apathetic tense depressed phobic panic | (p 1300) |
| Behavior | Normal compulsive ties | (p 130) |
| Speech | Normal aphonic defective | (p 386) |

health the nutrition and development the personality make up the body type and the posture decubitus and gait

Body Type—The body type rarely noted and observed furnishes considerable diagnostic information for the experienced clinician (Fig 1011)

Three main body types are recognizable (1) The linear asthenic or visceroptotic (2) the stocky or pyknic and (3) the normal type

The Linear or Slender Type—The linear, asthenic or visceroptotic individual commonly has a narrow head The eyes are close together The bridge of the nose the arch of the mouth and jaw are narrow and high The teeth are often crowded The bite is irregular and malocclus

more apt to occur than in the stocky type Psychiatrists recognize that from this group are recruited the *schizophrenics*

The Stocky Type—The stocky individual is usually slow growing The head is broad the skull brachycephalic The eyes are far apart the nasal bridge is broad and the lower jaw large and strongly defined Palatal

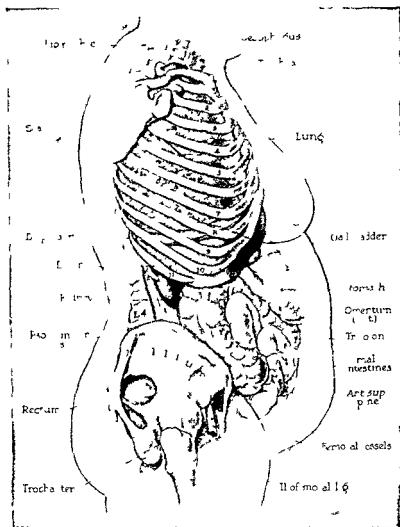


Fig 1013—Viscera in obese woman Right side

arches are broad and teeth are set far part The neck is apt to be short with the shoulders rounded and stooping

These individuals commonly have flattened lumbar curves The extremities and the muscles are short and thick and the trunk is long There is a tendency to *myopia* The stomach is of the steer horn type and the

Courtesy of S H Camp & Company

high Ptotic youngsters are reluctantly dragged by anxious mothers to the doctor's office for a tonic or something that will make the food stick to the ribs. In their sports and recreations the slender type excels in games of skill such as tennis and golf. The lanky tap dancer has all the common characteristics of this group.

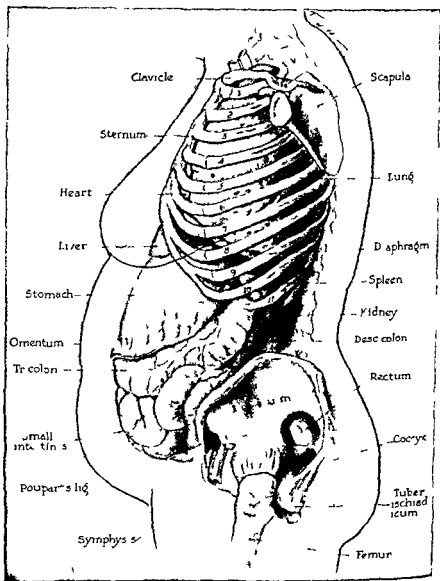


Fig 1012—Viscera in obese woman Left side *

CLINICAL DISTURBANCES OF THE SLENDER TYPE—The ptotic types tend to develop the *rheumatoid type of arthritis* rarely the hypertrophic type. *Gastroduodenal ulceration* is frequent but *gallbladder disease* is uncommon. *Sinusitis hyperopia* and *pulmonary and other respiratory diseases* are

Courtesy of S H Camp & Company

the breast bone forms the farthest part of the body anteriorly. The lower part of the abdomen is held flat. The back curve shows a slight dorsal kyphosis and gentle lumbar lordosis. The toes point forward so that the weight bearing line passes at all times through the center of the body and midway through the transverse arch of the foot.

Sitting Posture—Good sitting posture may be accomplished in a reasonably high broad article of furniture both feet are planted on the ground the buttocks and shoulders resting squarely against the back of the seat. Good sitting posture is virtually impossible in upholstered furniture the inviting easy chair any automobile or chairs in public carriers.

Reclining Posture—Good reclining posture requires a relatively firm mattress the head being supported by one small pillow or preferably none.

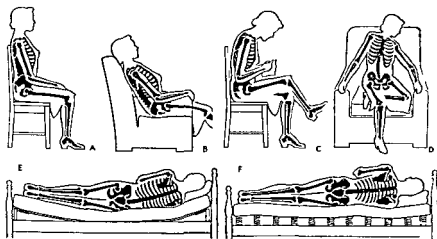


Fig 1015.—Sitting and sleeping postures. A Correct, and B C D incorrect sitting postures. E Illustrates the trouble that can be caused by a sagging mattress. F The best type of bed box springs or an innerspring mattress gives equal support to all parts of the body.*

Decubitus—In bed the position of the patient occasionally gives some indication of the diagnosis. The normal individual changes position frequently and is not averse to moving about. The afflicted patient occasionally assumes a fixed decubitus and objects to any alteration.

Gait—The normal gait is simply good standing posture in motion. Weight bearing is transferred forward on the outer side of the foot from talus to calcaneus. The forward foot is placed with the heel resting on the floor and the foot in dorsiflexion. The weight then rolls over the heel which acts as a fulcrum. The posterior leg muscles contract and bring the anterior part of the foot in contact with the floor while the heel is lifted. In this movement the foot acts as a lever the talus as the fulcrum. The flexor muscles of the toe contract and cause the toes to grip the floor and propel the body forward (heel and toe gait).

transverse colon usually occupies a position above the level of the umbilicus

CLINICAL DISTURBANCES OF THE STOCKY TYPE—The stocky patients tend to be *overweight*. They develop *hypertrophic osteo arthrosis*. In the women *gallbladder disease* is more frequent than peptic ulceration. The level of the blood pressure is higher. There is a tendency in later life to the development of *hypertension arteriosclerosis angina pectoris coronary thrombosis* and *cerebral hemorrhage*. The pulmonary difficulties are apt to be complicated by *emphysema*.

The Normal Type—The third or normal type is midway between the linear and stocky statures. No generalities are warranted. In fact it must

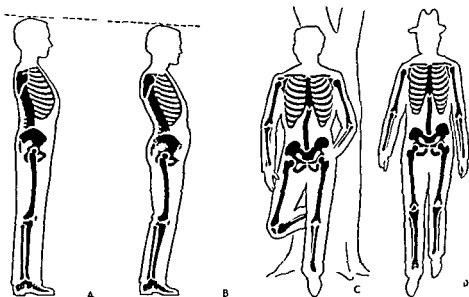


Fig 1014—Standing and walking postures. A Good standing posture. Note the straight weight bearing line from head through center of body and midway through transverse arch of foot. B Shows the commonest faults of poor posture resulting in weight swaying forward on legs. C Showing how pelvis is tilted and spine curved when weight is thrown on one leg. D Good walking posture. Note that body is erect as in good standing posture. Feet are parallel and close together and more weight is thrown on ball of foot than on heel.

be understood that the principles listed for the bodily types are broad and subject to many exceptions.

Posture—Abnormalities in body mechanics account for many of the skeletal difficulties encountered in office practice. It is exceptional to find notations concerning posture in medical records, particularly those of institutional medicine where the patient is often examined in bed.

Poor posture is almost universal. It causes a large proportion of the clinical complaints that daily confront the average practitioner. These include skeletal and joint pain (p 2802), *asthenia* (p 2890) and a fair number of gastro intestinal symptoms (p 1767).

Standing Posture—Good posture in the standing position consists in an erect position with the head up, chin in and the chest raised so that

the breast bone forms the farthest part of the body anteriorly. The lower part of the abdomen is held flat. The back curve shows a slight dorsal kyphosis and gentle lumbar lordosis. The toes point forward so that the weight bearing line passes at all times through the center of the body and midway through the transverse arch of the foot.

Sitting Posture—Good sitting posture may be accomplished in a reasonably high broad article of furniture both feet are planted on the ground the buttocks and shoulders resting squarely against the back of the seat. Good sitting posture is virtually impossible in upholstered furniture the inviting easy chair any automobile or chairs in public carriers.

Reclining Posture—Good reclining posture requires a relatively firm mattress the head being supported by one small pillow or preferably none.

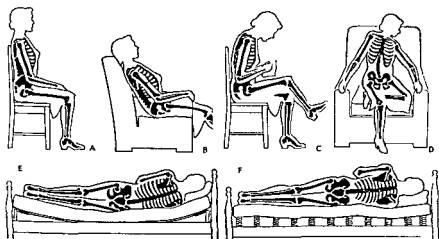


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DIFFERENTIAL DIAGNOSIS OF

Disturbances of Decubitus

Observation of the position of the patient, in bed, may suggest diagnostic possibilities for more exhaustive investigation

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

Immobility

Pain

With trauma or inflammation

Myxedema

Thickened skin obesity and low BMR. (p 1193) Note therapeutic response to thyroid extract

Adrenal Cortical Deficiency

Hypotension asthenia and pigmentation (p 1673) Note therapeutic response to saline

Psychiatric Disorders

Particularly neurasthenia (p 1350) and depressive psychoses (p 1368)

Paralysis Agitans

Mask like face Festinating gait (p 1505) Intention tremor relieved by belladonna

Forward Failure

Syncope (p 921) and shock (p 928) Associated with acute hypotension and profuse sweating

Poisoning

With hypnotics sedatives narcotics and anesthetics

Restlessness

Pain

Particularly colics

Hyperthyroidism

Goiter tachycardia exophthalmos and elevation of BMR (p 1197) Note therapeutic response to iodide

Psychoses

Particularly agitated varieties (p 1368)

Hemorrhage

Exsanguination unaccompanied by shock (p 928)

Poisoning

From cerebral stimulants particularly caffeine strychnine and amphetamine

Chorea

As part of rheumatic fever syndrome (p 186)

Opisthotonus

See p 3520

Orthopnea

See p 2016

Seated with Head Resting Forward on

Clasped Hands

Pericardial Effusion

Increased area of cardiac dulness Obliteration of cardiohepatic angle Diminished or absent apex impulse and pear shaped silhouette on fluoroscopy (p 795) Therapeutic response to salicylates in rheumatic fever Otherwise consider aspiration (p 852)

Triangle (Seated Resting Backward on Out stretched Hands)

Poliomyelitis

Stiffness of spine and peripheral motor palsies Clear cerebrospinal fluid containing lymphocytes increasing quantities of protein and a normal sugar content

Bronchial Asthma

Allergic disturbance with bronchospasm ballooning of chest generalized rales and prolonged aspiration Relieved by epinephrine (p 2101)

Unusual Attitudes

| | |
|-------------------------------------|---|
| Hysteria | Without demonstrable organic neurologic disease. Catalepsy (p. 1333) |
| Huntington's Chorea | Hereditary syndrome with persistent irregular movements, disturbances of speech, and mental deterioration (p. 1417) |
| Friedreich's Ataxia | Hereditary disturbance with athetosis, ataxia, and disturbances of speech (p. 1415) |
| Progressive Lenticular Degeneration | Athetosis, explosive laughter, and hepatic cirrhosis (p. 1418) |
| Muscle Dystrophies | Idiopathic disturbances of musculature. Usually associated with emacia and hypertrophy or atrophy of fibers (p. 2880) |



Fig. 1016.—Normal gait. Foot of Nordic man. Taken by Malvina Hoffman in Hall of Races of Mankind. (Courtesy of Field Museum of Natural History.)

Each pace approximates 18 inches. The toes point directly forward and the sound of the step is virtually inaudible. In walking with the heel of the forward foot on the floor, the knee is fully extended. As the heel leaves the floor, the knee is flexed. At this time the weight of the body is transferred wholly to the forward foot; the backward foot is clear of the floor and swings ahead of the forward foot. At no time during the normal pace should the entire foot rest flat on the floor. The gait should constitute a rolling motion from behind forward.

Normal gait is dependent upon factors other than mere integrity of the local structures and mechanics. It requires integration with cerebellar and vestibular mechanisms and with distant motor and sensory conditions.

DIFFERENTIAL DIAGNOSIS OF

Disturbances of Gait in the Adult

The normal gait is dependent for its integrity upon the coordinated functions of a variety of structures. These include peripheral sensorium and musculature labyrinthine and vestibular mechanisms and cerebral and cerebellar centers. In consequence disturbances in gait require intensive investigation for determination of the exact cause of the difficulty. Abnormalities in early life are separately discussed in the section devoted to Pediatrics (p 2736)

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | | |
|-------------------------------------|---|--|
| Astasia | | |
| Astasia abasia | | Inability to stand despite absence of evidences of organic disease. A manifestation of hysteria (p 1308) |
| Ataxia | | See p 3585 |
| Claudication (Interruption of Gait) | | |
| Peripheral Arteriosclerosis | | Usually in males after the 5th decade. Often in one leg. Arterial pulsations feeble or absent. Vessels visualized by x ray (p 1030) |
| Thrombo-angitis Obliterans | - | In Russian or Polish Jewish males before 5th decade. May involve both legs and arms. May be associated with multiple attacks of phlebitis. Intractable pain. Arterial pulsations diminished to absent (p 1029) |
| Raynaud's Disease | | In young females. Upper extremities usually bilaterally involved. Arterial pulsations unimpaired (p 1000) |
| Delay in Walking | | See p 2736 |
| Falling to One Side | | |
| Cerebellar Tumor | | Often associated with nystagmus, vertigo, tinnitus and unilateral deafness (p 1426). Refer to neurosurgeon. |
| Fetination | | |
| Paralysis Agitans | | Increasing tempo of gait with propulsion (lurching forward) and retropulsion (lurching backward). Mask like facies. Intention tremor relieved by belladonna (p 1505) |
| Limp | | |
| In childhood | | See p 2832 |
| In the adult | | |
| Osteoarthritis | | Metabolic disturbance in obese middle aged adults. Low BMR. X rays show narrowing of joint spaces, thickening of articular surfaces, lipping and spur formation (p 2855) |
| Rheumatoid (Atrophic) Arthritis | | In young adults with marked vasomotor disturbances and muscle atrophy. Often associated with fever and increased erythrocyte sedimentation time. X rays show narrowing of joint spaces, atrophy of articular surfaces and ankylosis (p 2910) |
| Rheumatic Fever | | Episodes of acute inflammation often with effusion. Associated with endocarditis, pericarditis and myocarditis. Specific response to salicylates (p 186) |

| | |
|------------------------------------|---|
| Infectious Arthritis | Pyogenic gonorrheal or tuberculous involvements of hips knees or ankles (p 2910) Seek focus Identify bacterial cause |
| Poliomyelitis | With muscle atrophy producing flail leg (p 457) Check history |
| Gout | Most frequent in obese middle aged males with involvement of great toe (podagra) ankles or knees Associated with increase in blood uric acid Urate deposits in lobes of ears (tophi) Specific response to colchicine (p 2867) |
| Low Back Strain | Mechanical defects often due to faulty posture acute injury or microtraumas (p 3072) Refer to orthopedist |
| Herniation of the Nucleus Pulposus | Acute back strain often manifested by sciatic syndrome and loss of tendon reflexes at knee and ankle (p 3074) Spinal fluid may be xanthochromic Refer to neurosurgeon |
| Foot Strain | Often the result of faulty mechanics and bad shoeing (p 3081) |
| Scissors Gait | |
| Little's Disease | Congenital spastic paralysis with hyperflexia clonus and Babinski sign (p 2948) |
| Mincing Gait (Small Steps) | |
| Senility | Particularly in debilitated patients beyond the age of 70 |
| Peripheral Arteriosclerosis | As above |
| Marie Strumpell Disease | Rheumatoid (atrophic) arthritis of the spine as above |
| Spasticity | |
| Little's Disease | See above |
| Multiple Sclerosis | Deminated neuropathy pursuing remittent course between 20th and 35th years Frequently manifested by diplopia pallor of temporal half of optic disk and hyperreflexia (p 1504) |
| Steppage Gait | |
| Tabes Dorsalis | With pupillary changes abert reflexes and positive tests for syphilis in blood and cerebrospinal fluid (p 1464) Treat intensively with penicillin |
| Peripheral Neuritis | Usually associated with sensory abnormalities of toxic or metabolic origin (p 1439) Remove cause Treat intensively with thiamine |
| Waddle | See p 2737 |
| Sid Wheel Gait | |
| Hemiplegia | Usually due to cerebral embolism or thrombosis with unilateral paralysis and hyperreflexia (p 1439) |

CHAPTER 157

THE PHYSICAL EXAMINATION TEGUMENTARY SYSTEM

THE topographical examination begins logically with inspection of the body surface. The tegumentary system is composed of the *skin* and its appendages *hair*, *nails* and *sweat* and *sebaceous glands*.

ANATOMIC REVIEW

The Skin—The skin affords protection against invading organisms and acclimatizes the individual to changes in external environment. It acts as a vast tactile surface and assists in heat regulation, water balance and the sexual reflexes. At the body orifices the skin is continuous with the mucous surfaces which share its qualities and exhibit alterations identical with dermatologic manifestations.

The color of the skin depends upon (1) the thickness of the horny layer which may vary from 0.5 mm in the eyelids to 4 or more mm in the palms and soles, (2) the pigment content of most importance in racial differentiation and (3) local vascular conditions relative to the extent of the capillary bed, the volume of the red blood corpuscles and their oxygen saturation. Thus local and transitory alterations in skin color are usually the result of capillary constriction or dilatation, anemia or polycythemia or a lessened concentration of oxyhemoglobin.

The skin consists of two layers, the *epidermis* and the *cutis*. The latter is composed of a corium and a layer of subcutaneous tissue which loosely attaches the skin to the underlying parts, thus affording a degree of mobility.

The Epidermis—The epidermis, cuticle or epithelial layer, the most superficial of the skin structures, is made up of five recognizable layers: (1) The stratum germinativum or basal-cell layer, (2) the stratum spinosum, prickle-cell or squamous-cell layer or rete mal pighi, (3) the stratum granulosum or keratohyalin layer, (4) the stratum lucidum and (5) the superficial stratum corneum or horny layer. The stratum germinativum and the stratum spinosum are often considered as one layer and called the rete mucosum.

The *basal cell layer* consists of a single row of vertically arranged, fairly regular cylindrical cells. Their lower surfaces are flattened and rest directly upon the cutis to which they are anchored by short protoplasmic processes, the rete pegs. Between these and the other cells of the epidermis, except those of the stratum corneum, are intercellular spaces traversed by numerous delicate protoplasmic fibrillae or strands. The cells of the basal layer (stratum germinativum) are the only structures of the epidermis normally endowed with reproductive powers. They are constantly dividing and forming new cells which are pushed upward successively becoming part of each epidermal stratum. A normal function of special cells of the basal layer is pigment production. The pigment granules are distinctly visible in the cell protoplasm about the nucleus. See *Epithelioma Basal Cell Type* (p. 3220).

The *stratum spinosum*, or prickle-cell layer, consists of four to eight rows of polygonal cells resting upon the basal-cell layer. The prickles or intercellular bridges are very well developed. The intercellular spaces also contain tissue fluid. Prickle cells reproduce only in certain pathologic states. See *Epithelioma Prickle Cell Type* (p. 3223).

The *stratum granulosum* comprises one or two layers of flattened cells rendered distinctive by the presence of rather large, deeply staining cytoplasmic granules (keratohyalin). The *stratum lucidum* is transparent and poorly delineated except in the palms and soles. The nuclei are shrunken and vestigial. The cells contain eleidin, whose chemistry is poorly understood but which is believed to be the precursor of keratin. The most superficial layer is the *stratum corneum* or horny layer. This consists of flattened cells, clear and transparent, non-nucleated, without intercellular bridges and composed of a resistant cornified keratin. See *Keratosis* (p. 3166).

The Cutis—The cutis comprises the corium, dermis or true skin and the

tissue. The *corium* is the fibrous and elastic mantle of the body and contains and supports the glandular structures the hairs the muscles the blood and lymphatic vessels and the nerve elements with their end-organs of touch and sensation. The major portion of the corium is occupied by connective tissue fibers (collagen) of white fibrous tissue which unite to form bundles. These bundles are parallel to the surface and are surrounded by a thick web of yellow elastic fibers. This firm and elastic network is most compact in the upper or papillary portion of the corium and less so in the lower or reticular portion.

The lowest part of the cutis the *subcutis subcutaneous tissue* or *panniculus adiposus* has a loose fibrous structure in which are contained large amounts of fatty tissue. The fat content varies with the individual and the region of the body. The fat cells are arranged in lobules supported and surrounded by a connective tissue framework. No fat cells are present in the lips scrotum or nose.

NERVES OF THE SKIN—The skin is the most important of the tactile organs and is richly supplied with sensory nerves. Medullated and nonmedullated fibers of the voluntary and involuntary nervous systems with their specialized nerve endings are present in profusion.

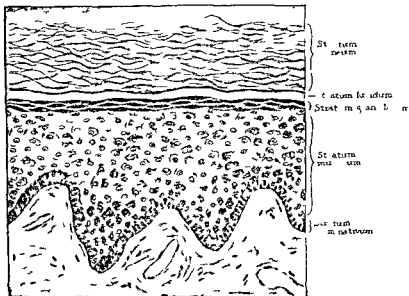


Fig. 1017.—Diagram illustrating the anatomy of the epidermis. Highly magnified.

The nerve fibers accompany the blood vessels of the subcutis and corium. Medullated fibers derived from the superficial nerve plexus run to the nerve papillae where they terminate in specialized end-organs as the corpuscles of Pacini and Meissner and the tactile bodies.

These are concerned with the perceptions of touch, heat, cold and pain.

Nonmedullated fibers arising from the involuntary nervous system are supplied to the sheaths of the blood vessels, the sweat glands and the arrectores pilorum muscles. These are under the control of the involuntary nervous system and function in the vasodermic phenomena such as blushing, flushing, gooseflesh and pallor.

VASODERMAL STRUCTURES—The cutaneous blood supply is abundant. It is important in heat regulation and water metabolism. The arterioles of the corium run vertically until they approach the epidermis where they turn horizontally, lose their muscular coats and give rise to capillary loops. In the living skin the capillary loops can be seen at the base of the finger nail under the low power of the microscope. The capillaries give rise to a rich plexus of subpapillary venous plexus which runs horizontally. In the digits there is a special type of anastomosis which functions in temperature regulation by shunting the blood.

The cutaneous circulation is controlled by the involuntary nervous system which can produce profound local and systemic changes in blood flow. The temperature and color of the skin are determined in part by vascular factors.

LYMPHATIC STRUCTURES—The lymphatic vessels are closely related to the plexuses of blood vessels in the corium and subcutis. There are numerous intercellular lymph spaces in the epidermis and the corium.

MUSCLES OF THE SKIN—Except for the voluntary muscle fibers found in the face and neck and concerned with facial expression, the muscles of the skin are all smooth or non-striated. The muscles that cause erection of the hair lie in close proximity to the adjacent or contiguous sebaceous gland. Sudden contractions of these erector muscles produce goose-flesh (*cutis anserina*) and in so doing they express the contents of the sebaceous glands. They also force blood deeply from the skin in this manner, preventing sudden excessive cooling. Smooth muscle fibers are also present about the sweat glands. Larger masses of smooth muscle fibers are arranged in isolated areas, such as the skin of the scrotum (*dartos muscle*) and in the areolae of the nipples.

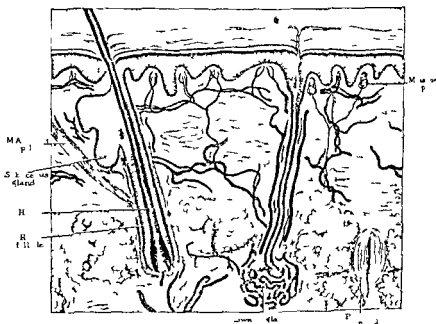


Fig 1018—Diagrammatic illustration of the anatomy of the skin and its appendages

PIGMENT OF THE SKIN—Melanin is elaborated by melanoblasts, specialized cells of the basal layer. Under normal conditions, most of the pigment is seen as granules in the cytoplasm of the basal cells. These pigment granules vary from a yellow to dark brown or black hue. They account for the distinctive racial differences, the so-called white, yellow, red, brown, and black races. The origin of melanin is in doubt. The generally accepted theory is that it is fabricated from materials conveyed to the melanoblasts by the blood stream.

The areolae and nipples, the genitalia and the circumanal regions are richly pigmented. Pigment is absent from the palms and soles.

The Hair—Hair covers the entire body except the palmar and plantar surfaces, the dorsa of the ungual phalanges, the glans penis, the inner surface of the prepuce, the labia minora and the mucosa of the lips. The *lanugo* or downy hair is implanted obliquely in the skin. It has no arrector pili muscle, but its sebaceous glands are very large. The *stiff hairs* or *bristles* occur in the eyelashes, eyebrows, and the nasal and auditory orifices. The bristles are implanted vertically. The *soft long hairs* occur on the cap, beard, mustache, axillae, pubes, and in the midline of the trunk.

The hair is divided into a portion which projects beyond the skin surface the *shaft* and a portion within the skin the *root* which rests in an epithelial invagination or hair follicle extending into the dermis and at times into the subcutaneous tissue. At the bottom of the follicle the hair bulb rests intimately upon the hair papilla which is of a structure similar to the vascular papilla of the corium.

To the hair follicles are attached the *arrectores pilorum muscles* which are nonstriated. They function to elevate the hair raising both the shaft and the surrounding skin above the surface to produce "goose flesh." The pigment of the hair is melanin.

The amount and distribution of body hair are determined chiefly by *endocrine* factors. They afford a gross index of sexual development (p. 2490). The infant is covered with lanugo. The scalp lanugo is shed after birth and replaced by new hairs that grow actively at this rate. In the female at puberty coarse hairs appear in the axillary and pubic regions. In the latter situation the upper border of the hair line is transverse. A considerable growth of hair may appear on the upper lip chin and chest, at the time of the menopause. In the male at puberty a discernible growth of hair occurs in the axillary and pubic regions on the beard the upper lip and over the sternum. The upper border of the pubic hair is triangular with the apex at the umbilicus.

The Nails—The finger and toe nails are specialized structures derived from the epidermis. They correspond to the stratum corneum except that they are harder and firmer.

The nail consists of the *body* or uncovered portion and the *root* or the portion embedded in the corium. The nail rests upon the nail bed. Part of the nail bed lies beneath the body of the nail, the remainder or matrix beneath the root. The whitish lunula marks the distal limit of the matrix. The nail grows from the matrix and pushes forward requiring four to nine months to complete its growth.

Below the matrix is the *subungual space* where abscess formation presents difficulties (p. 3973). Above the nail matrix and continuous with the lateral walls is the *paronychia* space formed by the outer and invaginated epidermis. It is the site of the common *felon* or "runaround." The skin overlying the base of the nail is termed the *eponychia*.

The texture of the nails and the color of the nail bed are noted. Small ungual hemorrhages (p. 3153) pigmented spots and capillary pulsation are sought.

The Sweat Gland—The sweat glands are located over the entire body except the lips glans penis and inner surface of the prepuce. They are especially numerous in the palms and soles. Two varieties the apocrine and eccrine are recognized.

The *apocrine* sweat glands are vestigial organs of scent most highly developed in lower animals. They are located in the axillae about the nipples and in the anogenital regions. They are developed from hair papillae and usually open into the hair follicle. The *mammary glands* are modified apocrine glands. In lower mammals the secretions from the apocrine glands increases during heat and the odor attracts the opposite sex. The *eccrine* sweat glands are coiled tubular structures with a simple cuboidal epithelium in the gland itself and a stratified epithelium in the duct which extends directly to the skin surface. The glands are located mostly in the subcutaneous tissue and at times in the depths of the corium. The duct opens on the surface at the sweat pore.

The sweat glands are under the control of the involuntary nervous system. As the result of sweat gland activity the normal skin is slightly moist. The degree of dryness or moistness varies widely depending on the temperature of the body and the environmental temperature and humidity. In examining the skin the *degree of sweating* should be evaluated with reference to these factors. The extent of distribution of the sweat is also of clinical importance. Symmetrical areas of the body should perspire equally. A local increase or decrease in sweat gland activity is commonly due to a neurological defect.

The Sebaceous Glands—The majority of the sebaceous glands are in direct association with the hairs the ducts opening into the hair follicle. They are present in nonhairy regions like the lips glans penis and mucous surface of the prepuce and the labia minora where sweat glands are lacking. There are no sebaceous glands in the palms and soles. The largest glands are seen on the face (cheeks and nose) the scrotum labia majora, about the anus the Meibomian glands the acola of the nipple and the mons veneris. The scalp is richly supplied.

Sebum is not a true secretion but rather the fatty material liberated from cells which have degenerated. It also contains cast-off epithelial cells and detritus. In the absence of a patent duct a sebaceous cyst or wen (p. 308) is produced. Clogging of the duct with sebum and extraneous material gives rise to the familiar blackhead or *comedo*.

CLINICAL EXAMINATION OF THE TEGUMENTARY SYSTEM

In the course of the routine physical examination the practitioner observes but does not necessarily comment on the normal appearance of the tegumentary system. Deviations from the norm and particularly the presence of eruptions have great interest and significance. The latter require special investigation; they may represent local processes but often are manifestations of a systemic disorder. It is in this latter sense that clinicians refer to the skin as the 'mirror of the body'.

See *Atlas of Dermatology* p 3105 Table of *Differential Diagnosis of Presenting Dermatoses* (p 3110)

CHAPTER 158

THE PHYSICAL EXAMINATION HEAD AND NECK

THE CRANIUM

SIZE AND SHAPE

At birth the circumference of the head measures 14 inches. During the first year it increases $\frac{1}{2}$ inch during each of the first six months and $\frac{1}{4}$ inch during each of the second six months. At the end of the first year the total increase approximates 5 inches, the normal measurement approaching 20 inches. The subsequent rate of growth is slower. The circumference increases about 1 inch during the second year and $\frac{1}{2}$ inch a year for the next three years (from two to five years). From the end of the fifth year until the onset of adolescence there is an increase of $\frac{1}{4}$ inch each five years. See *Differential Diagnosis of Cranium in Infancy* (p. 2774).

The head measurement of the normal adult male averages 22 $\frac{1}{2}$ inches corresponding to a size 7 hat. The average circumference of the female cranium is 22 inches. In normal individuals the size of the head varies within narrow limits.

The head is irregularly oval. After protracted labor the infant usually presents an elongated misshapen head, the result of moulding before delivery. This deformity usually disappears by the end of the first month. During the first year the head may become asymmetrical as the result of lying too much in one position.

THE FONTANELLES

In the normal infant prominent features of the skull are the *anterior* and *posterior fontanelles*. The latter is the smaller and is formed by the junction of the parietal and occipital bones. It closes at about the second month of life. The *anterior fontanelle* is formed at the junction of the frontal and parietal bones. It decreases gradually in size and should be completely closed by the end of the second year. The fontanelles may show deviations in closure; they may bulge or exhibit depression depending upon intracranial and intraventricular pressure relationships. See *Fontanelles Disturbances of* (p. 2729).

The two fontanelles are joined by the *sagittal suture*. Beneath the suture is the *superior longitudinal sinus*. Before closure of the fontanelles this sinus may be entered for diagnostic venipuncture or intravenous injection.

The lateral angles of the anterior fontanelle overlie the *lateral ventricles of the brain*. Aspiration of the ventricle may be performed at this site before the fontanelle has closed (p. 3783).

THE VAULT OF THE SKULL

With the closure of the fontanelles the vault of the skull assumes adult configuration. Several landmarks are of clinical importance. The *external*

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See *Atlas of Dermatology* p 3105 Table of *Differential Diagnosis of Presenting Dermatoses* (p 3110)

occipital protuberance is situated in the midline posteriorly. *Cisternal puncture* (p 3783) is performed just below this point. The *superior nuchal line* runs laterally on each side from the external occipital protuberance and can be followed forward to the lateral portion of the temporal bone. This marks the course of the lateral sinus.

The *mastoid process* containing the mastoid cells is palpable behind the ear. The anterior and posterior borders, the apex and external surface

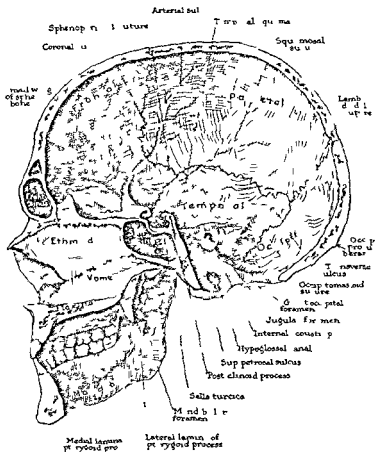


Fig 1019—Med an section of skull with mandible viewed from left

of this process are all available for superficial examination. See *Mastoiditis* (p 2146).

The lateral aspects of the vault of the skull are formed posteriorly by the *parietal eminences* and anteriorly by the *frontal eminences*. Below the frontal eminence is the *superciliary ridge* which indicates the position of the frontal sinus.

THE SCALP

The vertex is covered by the scalp and hair. The amount, texture and color of the hair show great individual variations in health and may be

DIFFERENTIAL DIAGNOSIS OF

Disturbances of the Head in Adults

Disturbances of the head are much more frequently observed in infancy than in later life. The child's head suffers from trauma during its passage through the birth canal and it presents abnormalities due to congenital anomalies, metabolic derangements and glandular dystrophies. Many of these have ominous significance as discussed in the section devoted to Pediatrics (p. 2723).

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

Enlargement

Acromegaly

Anterior pituitary deficiency. Beeting of brows. Prognathus. Spade hands and feet. X-ray changes in sella turcica (p. 1156).

Leontiasis Ossium

Osseous abnormality producing lion head (p. 2878).

Osteitis Deformans

Paget's disease of skull. Enlargement of cranium, progressive kyphosis and bowing of tibiae. Specific cotton wool changes revealed by x-rays (p. 2879).

Hydrocephalus

With or without neurologic disturbances and impairment of intellect (p. 2774).

Dermatoses of Scalp

See p. 3254.

Headache

See p. 1510.

Isolated Irregularities

Hematoma of Scalp

Following trauma. Exclude fracture by x-ray (p. 1450).

Fracture of Skull

Requires confirmation by x-ray. Consult with specialist.

Herniation of Brain

Following surgical decompression.

Dermoids

Near orbit, glabella or external angular region of frontal bone.

Benign Tumors

Particularly sebaceous cysts, lipomas and osteomas. Excision may be difficult.

Malignant Neoplasms

Metastatic carcinomatosis (p. 572). Multiple myeloma with Bence Jones proteosuria and characteristic cells in bone marrow smears (p. 1035).

Cellulitis and Phlegmon of Scalp

With widespread edema. Hazard of intracranial complications.

Tertiary Syphilis

Gummas. Therapeutic response to iodides (p. 3286). Serologic findings may be inconclusive.

Prominence of Veins

With alopecia (p. 3439) or osteitis deformans (p. 2879).

Tortuosity of Arteries

With generalized arteriosclerosis (p. 981) and localized temporal arteritis (p. 1510).

Tenderness

Cellulitis and Phlegmon

See above.

Overlying Brain Abscess or Tumor

Evidences of increased intracranial pressure (p. 1468). Localized neurologic manifestations (p. 1423). Consult neurosurgeon before "pans" tap.

Cervico-occipital Neuralgia

Usually radiating from base of skull along path of nerve. Often associated with induration of fascia and muscles. Relieved by pressure.

| | |
|-----------------------------|--|
| Dark Pigmentation | Pregnancy (chloasma gravidarum) adrenal cortical deficiency plumbism, argyria pellagra hemochromatosis hyperthyroidism, chronic arsenic poisoning ochronosis and Hodgkin's disease (pp 3154-3242) |
| Ruddiness (Rubor) | Normal complexion. May be associated with exposure to wind and sun backward failure polycythemia vera mitral stenosis emphysema pituitary basophilism, hypertension and alcoholism Check hemogram, hematocrit, blood pressures and x rays of chest and skull. |
| Malar Flush | Pulmonary tuberculosis myxedema congenital cardiac disease mitral stenosis and lobar pneumonia Check B.M.P. chest x ray and ECG |
| Dermatoses of Face | See p 3266 |
| Neuromuscular Changes | |
| Facial Asymmetry | Congenital facial hemiatrophy Facial palsy and tumors involving soft tissues Check neurologic status |
| Facial Tics | Habit spasm May be associated with chorea tic douloureux, hyperthyroidism and Friedreich's ataxia |
| Peripheral Facial Paralysis | Bell's palsy with inability to wrinkle forehead (p 1484) |
| Central Facial Paralysis | Ability to wrinkle forehead maintained May be associated with cerebral hemorrhage thrombosis tumor or abscess Also occurs with mastoiditis petrositis and labyrinthitis May accompany general paresis multiple sclerosis encephalitis bulbar poliomyelitis cerebrospinal meningitis trauma or surgical procedures Check neurologic status Refer to neurologist |
| Pain in Face | See p 2132 |
| Edema | |
| Of Eyelids | May be associated with dissipation, infections of eye or orbit, cavernous sinus thrombosis trichinosis nephritis nephrosis or allergy particularly dermatitis from nail polish Examine eyes sinuses and urine Check blood culture and hemogram. |
| Of Face | With sunburn, iodism, nephrosis and nephritis Following measles and smallpox. |

characteristically affected by disease (p 3439) The scalp veins are quite prominent in infancy particularly when the child cries In an emergency they may be used for intravenous medication infusion or transfusion (p 3773) In the adult the temporal vessels may be exceedingly tortuous and prominent and in rare instances diseased

The scalp consists of five layers Retraction of the tough fibrous septa results in severe hemorrhage when vessels are divided Emissary scalp veins drain into the intracranial venous sinuses Due to the many anastomoses between the veins of the scalp and the intracranial venous sinuses

DIFFERENTIAL DIAGNOSIS OF

Abnormalities of Face

From facial appearances it is permissible occasionally to draw inferences concerning physiologic and pathologic disturbances. Enthusiastic endocrinologists have a tendency to transcend facts by drawing unwarranted conclusions concerning glandular function when the patient presents some of the lesser features of a full blown hormonal syndrome. Experienced clinicians by contrast, recognize that these small signs are often familial traits of little clinical importance.

In conjunction with observations noted in the chart below the practitioner is referred also to additional material dealing with facies (p 3509) eye (p 1521) ear (p 2113) nose (p 2110) mouth (p 1668) jaw (1705) and salivary glands (p 3517).

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

Changes in Contour

Moon Face

Obesity Myxedema with low B.M.R. (p 1193) Eunuchism and Frohlich syndrome—with hypogonadism (p 1166)

Oval Face

Pituitary habitus Malnutrition Giantism (p 1155) Hyperthyroidism with elevation of B.M.R. (p 1197) Adrenal cortical or anterior pituitary deficiency (p 1673)

Bulldog Face

Achondroplasia with dwarfism (p 2762)

Buffalo Head

Pituitary basophilism with hypertension, vinitism and striae (p 1159)

Lion Face

Leontiasis ossium (p 2878) Osteitis deformans (p 2879) Leprosy with anesthetic nodules (p 273)

Bird Face

Congenital ankylosis of jaw (p 2810)

Mongolism

Mongolian idiocy (p 1165)

Beetle Brow with Prognathus

Acromegaly (p 1156)

Prominence of Forehead

Rickets (p 2850) Hydrocephalus (p 1409) Neonatal syphilis with positive serology (p 2787)

Virilism in the Female

Familial May accompany menopause pituitary basophilism adrenal cortical tumors and arrhenoblastomas (p 2575)

Feminization in the Male

Familial May accompany senility hypopituitarism and hypogonadism (p 2481)

Shaggy Eyebrows

Acromegaly (p 1156)

Scant or Absent Eyebrows

Myxedema with low B.M.R. (p 1193) Leprosy with anesthetic nodules and thickened nerve trunks (p 273)

Changes in Color

Pallor

Normally sallow complexion May be associated with anoxia anemia hemorrhage for ward failure senility arteriosclerosis malignancy chronic invalidism chronic infection, cardionephritis aortic insufficiency subacute bacterial endocarditis and lead poisoning Check hemogram urinalysis and blood culture

Green Tint

Chlorosis (p 1088) Get hemogram

Cafe au lait

Subacute bacterial endocarditis (p 286) Generalized arteriosclerosis and nephritis Check hemogram urinalysis and blood culture

THE NECK

The neck is tightly packed with numerous muscles viscera large blood vessels and important nerves. The various structures are separated by

 DIFFERENTIAL DIAGNOSIS OF

Facies

Changes in facial expression during the course of illness often furnish prognostic clues. The practitioner on entering the sickroom may note at a glance that the patient appears better or worse. Looking good is usually the result of an alertness of the eyes and a general appearance of brightness; it must not be confused with gleaming eyes and unjustified euphoria frequently seen in hyperthyroidism, severe influenza and tuberculosis. The more ominous facial expressions are those due to acute anemia, syncope or shock. They reach their full development in the hippocratic syndrome when the face appears pinched, the nose prominent, the eyes sunken, the brow cold and clammy and the color that of an ashen cyanosis. Such a clinical picture is almost an invariable sign of impending difficulty.

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|------------------------|---|
| Hippocratic | Generalized peritonitis (p 1923) with associated forward failure and overwhelming infection. Consult surgeon. |
| Mask like | Idiocy (p 1332). Myxedema or cretinism with low B.M.R. (p 1193). Scleroderma with hide bound skin (p 3427). Paralysis agitans with intention tremor (p 1505). Myasthenia gravis with therapeutic response to neostigmine (p 2886). Mental depression. Nasal obstruction particularly due to adenoids. |
| Strained | Hyperthyroidism with elevated B.M.R. (p 1197). Bronchial asthma (p 2101). Manic states. Strychnine poisoning (risus sardonicus) (p 3869). Tetanus (p 294). Lenticular degeneration with uncontrolled laughter (p 1418). |
| Progeria | Premature senile expression with pituitary infantilism (p 1164). |
| Wax like Face | Myxedema or cretinism with low B.M.R. (p 1193). Lethargic encephalitis (p 449). Bulbar palsy (p 1504). Pernicious anemia with hyperchromic anemia (p 1077). Check neurologic status and hemogram. |
| Ivory Face | Congenital cardiac disease with murmurs (p 956). Myxedema with low B.M.R. (p 1193). |
| Hide bound Face | Scleroderma (p 3427). |
| Thickening of Features | Myxedema and cretinism (p 1193). Acromegaly (p 1156). Leprosy with anesthetic nodules (p 273). |

fascial planes which are continuous with those of the mediastinum and axilla. A knowledge of the anatomy of the neck is essential to a correct evaluation of physical findings and in understanding the mode of progression of pathological processes in the cervical region.

severe intracranial complications may result from seemingly inconsequential scalp infections

See *Scalp Disturbances of* (p 3254)

THE FACE

ANATOMIC REVIEW

The forehead with the *frontal eminences* laterally forms the upper margin of the face. The *superciliary arches* indicate the position of the *frontal sinuses*. Beneath the arch is the eyebrow which overlies the *superciliary ridge*. Situated between and connecting the superciliary ridges is a smooth somewhat triangular area the *glabella* below which the *fronto-nasal suture* can be felt as a slight depression at the root of the nose. The nasal bones, covered by soft tissues can be traced to their junctions with the nasal cartilages. On either side of the nasal bone the complete outline of the orbital margin can be made out. The contents of the orbits are elsewhere discussed (p 3619). Below and lateral to the orbit is the *zygomatic bone* which forms the prominence of the cheek. Beneath this is the *maxillary sinus* (p 3593). The nose accessory sinuses and oropharynx are more completely examined in the dark room (p 3595).

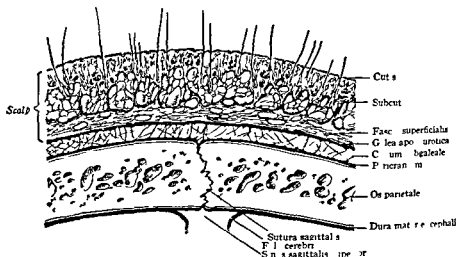


Fig 1000—Frontal section through the extracranial soft parts and the parietal bones

The lower margin of the face is formed by the *mandible* which may be palpated throughout its entire length. The *temporomandibular articulation* is quite superficial. Its position can be ascertained by defining the condyle of the mandible. When the mouth opens the condyle advances out of the *mandibular cavity* onto the articular tubercle. A depression is felt in the situation of the joint. See *Temporomandibular Joint* Description of (p 2965).

The *masseter muscle* imparts fullness to the angle of the jaw. The anterior margin of this muscle marks the course of the important *external maxillary artery* which makes its way upward toward the nose. Injury to this vessel produces brisk hemorrhage.

The face is innervated by the *fifth* (trigeminal) and *seventh* (facial) cranial nerves. The *trigeminal nerve* is sensory for the skin of the face, mucous membranes of the oral cavity and nasal sinuses and the anterior two thirds of the tongue. Its motor division supplies the muscles of mastication. The branches of the fifth nerve (ophthalmic, maxillary and mandibular) emerge through fissures and foramina in the cranial and facial bones. The *facial nerve* innervates the muscles of facial expression. It also supplies sensory fibers (taste) to the anterior two thirds of the tongue. The motor division is in close proximity to the internal and middle ear and leaves the skull via the stylomastoid foramen.

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| Hide bound Face | Scleroderma (p 3427) |
| Thickening of Features | Myxedema and cretinism (p 1193) Acromegaly (p 1150) Leprosy with anesthetic nodules (p 273) |

fascial planes which are continuous with those of the mediastinum and axilla A knowledge of the anatomy of the neck is essential to a correct evaluation of physical findings and in understanding the mode of progression of pathological processes in the cervical region

severe intracranial complications may result from seemingly inconsequential scalp infections

See *Scalp Disturbances of* (p 3254)

THE FACE

ANATOMIC REVIEW

The forehead with the *frontal eminences* laterally forms the upper margin of the face. The *superciliary arches* indicate the position of the *frontal sinuses*. Beneath the arch is the eyebrow which overlies the *superciliary ridge*. Situated between and connecting the superciliary ridges is a smooth somewhat triangular area the *glabella* below which the *fronto-nasal suture* can be felt as a slight depression at the root of the nose. The nasal bones, covered by soft tissues, can be traced to their junctions with the nasal cartilages. On either side of the nasal bone the complete outline of the orbital margin can be made out. The contents of the orbits are elsewhere discussed (p 3619). Below and lateral to the orbit is the *zygomatic bone* which forms the prominence of the cheek. Beneath this is the *maxillary sinus* (p 3393). The nose, accessory sinuses and oropharynx are more completely examined in the dark room (p 3395).

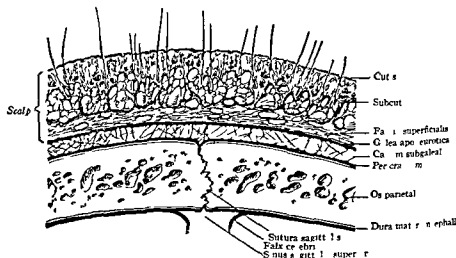


Fig 1090—Frontal section through the extracranial soft parts and the parietal bones

The lower margin of the face is formed by the *mandible* which may be palpated throughout its entire length. The *temporomandibular articulation* is quite superficial. Its position can be ascertained by defining the condyle of the mandible. When the mouth opens, the condyle advances out of the *mandibular cavity* onto the articular tubercle. A depression is felt in the situation of the joint. See *Temporomandibular Joint Dislocation of* (p 2263).

The *masseter muscle* imparts fullness to the angle of the jaw. The anterior margin of this muscle marks the course of the important *external maxillary artery* which makes its way upward toward the nose. Injury to this vessel produces brisk hemorrhage.

The face is innervated by the *fifth* (trigeminal) and *seventh* (facial) cranial nerves. The *trigeminal nerve* is sensory for the skin of the face, mucous membranes of the oral cavity and nasal sinuses and the anterior two thirds of the tongue. Its motor division supplies the muscles of mastication. The branches of the fifth nerve (ophthalmic, maxillary and mandibular) emerge through fissures and foramina in the cranial and facial bones. The *facial nerve* innervates the muscles of facial expression. It also supplies sensory fibers (taste) to the anterior two thirds of the tongue. The motor division is in close proximity to the internal and middle ear and leaves the skull via the stylomastoid foramen.

ANATOMIC REVIEW

Posteriorly the neck is formed by the cervical spine and is invested by the anterior and posterior spinal muscles. The movements of the neck are *flexion* approximating the chin to the chest, *extension* raising the chin to an angle of about 170 degrees, *lateral rotation* right to left approximating 60 degrees, and a *lateral motion* the lobe of the ear touching the shoulder.

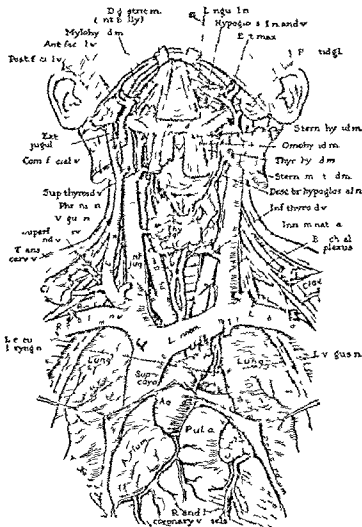


Fig. 1021.—D section of the anterior aspect of the neck and mediastinum after removal of the sternum, portions of both clavicles and many of the muscles. (Modified from Sobotta.)

Triangles of the Neck.—The front of the neck is subdivided by the sternomastoid muscle into anterior and posterior triangles.

The posterior triangle is bounded in front by the sternomastoid, behind by the trapezius and below by the clavicle. This triangle contains portions of the *subclavian vessels*, part of the *brachial plexus* and the *posterior cervical lymph node*. The pulsation of the sub-

DIFFERENTIAL DIAGNOSIS OF

Abnormalities of the Neck

Symptoms referable to the neck require intensive investigation because of the concentration of many important anatomic structures in this region. Some complaints such as pain and stiffness (p 3520) may represent disturbance in tissues as distant as meninges, vertebrae, pleura, gallbladder or heart.

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|---|---|
| <i>Cervical Lymphadenopathy</i> | See p 3518 |
| <i>Congenital Malformations</i> | |
| Port wine Nevus | Usually at nape of neck (p 3200) |
| Cervical Aunicle | Usually just below normal ear (p 2043) |
| Cysts and Fistulas | Branchiogenic antero-laterally Thyroglossal in median line |
| Torticollis | Wry neck (p 2816) Refer to surgeon for section of sternomastoid |
| Cervical Rib | Usually asymptomatic (p 2953) May be associated with scalenus anticus syndrome (p 2953) |
| Klippel Feil Syndrome | Ankylosis and shortening of neck (p 2817) |
| <i>Dermatoses</i> | See p 3254 |
| <i>Swellings and Tumors</i> | See p 3514 |
| <i>Trauma and Mechanical Disturbances</i> | |
| Fracture Dislocation | May be due to direct or indirect force. Confirm by anteroposterior and lateral x rays (p 3005) |
| Subluxation of Posterior Articular Facets | Productive of familiar "crick in neck". Relieved by traction and manipulation (p 2968). Confirm by lateral oblique x rays |
| Hematoma of Sternomastoid | Frequently present at birth |
| Scalenus Anticus Syndrome | Often associated with cervical rib. Associated with pains in arm due to pressure on brachial plexus and subclavian artery (p 2953). Relieved by surgery |
| <i>Vascular Disturbances</i> | See p 3516 |
| <i>Inflammations and Infections</i> | |
| Pyoderms | Particularly furuncles, carbuncles, pustular folliculitis and impetigo contagiosa (p 3248). Start antibiotic therapy |
| Cellulitis and Phlegmon | Usually associated with carbuncles (p 3249). Intensive antibiotic therapy |
| Tuberculous Lymphadenitis | Indolent inflammation (scrofula) often associated with persistent sinus and scarring. Diagnose by biopsy (p 3262) |
| Actinomycosis | Chronic inflammation often associated with persistent fistula. Organisms demonstrable in pus or tissue (p 489) |
| Ludwig's Angina | Cellulitis of floor of mouth (p 1708). Intensive antibiotic therapy supplemented by surgery |

THE THYROID GLAND

The thyroid gland is situated over the lower part of the larynx and the upper five or six rings of the trachea. It consists of *two lateral lobes* connected by an *isthmus* which lies on the second, third and fourth tracheal rings. A third or *pyramidal lobe* sometimes arises from the isthmus and extends upward toward the base of the tongue. The lateral lobes are

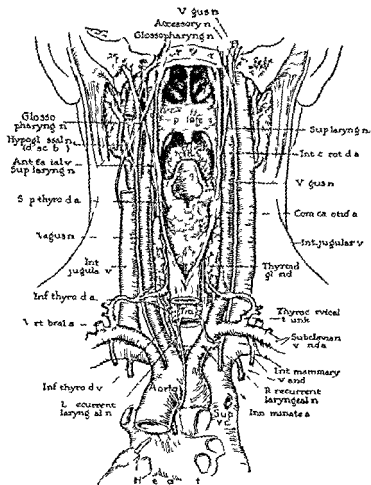


Fig. 10.—Posterior aspect of pharynx and larynx in relation to the great blood vessels of the neck and upper thorax.

conical, each lobe being about 2 inches long, $1\frac{3}{4}$ inches wide and about $\frac{3}{4}$ inch thick. The isthmus measures about $\frac{1}{2}$ inch in breadth and is approximately the same in depth.

Examination of the Thyroid Gland.—The thyroid gland is examined by palpation and auscultation. It is of the utmost significance to delineate accurately its size, shape and consistency. Palpation may be performed

clavian artery is palpable just above the clavicle. It is at this site that the *scalenus anticus muscle* may compress the vessel and impinge upon the plexus. See *Scalenus Anticus Syndrome* (p. 2933).

The *anterior cervical triangle* is bounded laterally by the sternomastoid muscle above by the imaginary line joining the mastoid tip to the lower border of the mandible. In the median line the two anterior triangles impinge.

Mesially the anterior triangles contain from behind forward (1) the *pharynx* (2) the *esophagus* (3) the *larynx* (4) the *trachea* (5) the *thyroid* and *parathyroid glands* and (6) the *carotid sheath*.

THE LARYNX AND TRACHEA

The cartilages of the larynx can be distinguished in the median line. The hyoid bone is easily palpable. A finger's breadth below is the *Adam's*

DIFFERENTIAL DIAGNOSIS OF

Disturbances of the Trachea

Disturbances of the upper rings of the trachea are easily recognized by inspection and palpation. Within the thorax the course of the windpipe is traced through its radiologic lucency. Primary lesions of the trachea are rare, but abnormalities due to impingement by contiguous structures are frequently encountered.

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

Deviation

Ipsilateral

Pulmonary fibrosis or atelectasis. Obtain x rays of chest.

Contralateral

Hydrothorax or pneumothorax. Tumors of pleura, lung, thyroid or mediastinum. Aortic aneurysm. Confirm physical signs with x rays. Diagnostic thoracentesis when indicated (p. 2030).

Tracheal Tug

Dilatation or aneurysm of aorta. Mediastinal tumor. Confirm physical signs by x rays.

Tracheal Compression

Enlargements of thyroid. Dilatation or aneurysm of aorta. Inflammations or neoplasms of mediastinum. Confirm physical signs by x rays.

apple which represents the prominence the upper borders and the two wings of the thyroid cartilage. Below the *Adam's apple* is a depression corresponding to the cricothyroid space. The operation of *laryngotomy* is performed at this site. The lower border of the depression is formed by the anterior ring of the cricoid cartilage. Below this the trachea and thyroid isthmus are felt.

The visual examination of the interior of the larynx is discussed in the chapter on dark room technic (p. 3604).

Tracheal Tug—With aneurysm of the arch of the aorta the trachea transmits the arterial pulsations. The 'tracheal tug' is noted by standing behind the patient, the thumb and forefinger of the examiner gripping the trachea while the patient tilts his head upward. When the sign is present there will be felt a downward pull synchronous with the cardiac systole.

Vascular

Subclavian artery

Pulsating tumor due to cervical rib or aneurysm (p 3576)

Aortic aneurysm

Causing episternal pulsation. Confirm by x ray of chest and serologic tests (p 968)

from behind or in front. When examining from behind the examiner presses his fingers over the side of the trachea the gland moving upward and downward with the act of swallowing.

The examination from the front yields considerably more information. The patient relaxes the sternomastoid muscle by approximating the chin to the chest. The examiner pinches the lobe between thumb and index finger determining the size as well as the consistency of the gland. For examination of the right lobe the examiner with his right hand pushes the trachea from left to right. With the thumb and fingers of his left hand he pinches the right lobe throughout its length to determine its height, width, depth, and consistency. Reversing procedures the left lobe is examined. The isthmus is palpated directly over the tracheal rings. A substernal extension is sought by mapping out the lower border the patient extending the chin while the examining finger is pressed in the suprasternal notch.

Normally the blood flow in the thyroid gland is not detectable. Under pathological conditions a *thrill* may be felt and *murmurs* auscultated in the region of the superior thyroid artery.

See *Clinical Disorders of the Thyroid* (p 1191) *Differential Diagnosis of Increased* (p 720) and *Decreased* (p 719) *Metabolic Rate*.

THE PARATHYROID GLANDS

These glands usually four in number are situated behind the lateral thyroid lobes, two on each side. The average weight of a single gland in an adult is in the neighborhood of 30 mg. At operation the parathyroid glands are apt to be confused with small collections of fat, lymph nodes, or nodules of thyroid tissue.

Aberrant parathyroids are not uncommon and they may occur within the tissue of the thyroid, behind the esophagus, at the base of the tongue, or in the anterior or posterior mediastinum. Even when enormously enlarged the glands are rarely palpable.

See *The Parathyroids* (p 1223) *Differential Diagnosis of Hypercalcemia* (p 723) and *Hypocalcemia* (p 724).

THE CAROTID SHEATHS

Lateral to the trachea and esophagus in the anterior triangles of the neck are the carotid sheaths. These are fascial tubes which enclose (1) the *common carotid arteries*, (2) the *internal jugular veins*, (3) the *vagi*, and (4) the *hypoglossal nerves*. Opposite the upper border of the thyroid cartilage the common carotids divide into external and internal branches.

Above and behind the angle of carotid bifurcation lies the *carotid body*, a small nodule of chromaffin tissue. Just before the bifurcation the vessel

DIFFERENTIAL DIAGNOSIS OF

Swellings and Tumors of the Neck

In addition to swellings due to cervical lymphadenopathy (p 3518) the neck may be the site of tumors of congenital or acquired origin. The ease with which the mass may be palpated facilitates investigation and usually clarifies the diagnosis without need for further aids.

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

Subcutaneous Tissues

Congenital Anomalies

Cystic hygroma in anterolateral region. Dermoid in median line. Branchial thyroglossal cyst often with fistula in anterolateral region (p 1218). Refer to surgeon.

Neoplasms

Lipomas and fibromas usually situated posteriorly. Refer to surgeon.

Metabolic Disturbances

Supraclavicular fat pads in cretinism. Note low BMR.

Infection

Of deep fascial planes (p 3519). Of pre-existent congenital cysts. Treat conservatively unless airway is compromised.

Thyroid Gland

Cysts and Neoplasms

Thyroidal or thyroglossal. Localized and diffuse adenomas (p 1221). Carcinoma (p 1221). Consider surgery.

Goiter

Hyperplastic (soft and uniform). Colloidal (firm and uniform) (p 1198). Get BMR. Note response to iodide.

Inflammatory

Thyroiditis and strumitis (p 1221). With acute inflammatory signs and tenderness. Treat conservatively unless airway is compromised.

Vascular

Thyroid apoplexy particularly in colloidal goiters. Acute pain and swelling (p 1208).

Parathyroid and Juxta-Parathyroid Glands

Neoplasm

Irregular and globular tumors just above and behind pole of thyroid (p 1233). May cause hyperparathyroidism with elevation of serum calcium, diffuse osteoporosis and multiple bone cysts demonstrable by x-rays. Refer to surgeon.

Sternomastoid

Hematomas

Usually congenital (p 2775).

Meninges

Meningocele

In median line posteriorly (p 2772).

Carotid Bodies

Neoplasm

At bifurcation of carotid artery (p 922). Pressure may cause syncope from hypersensitivity.

Salivary Glands

See p 3518.

Esophagus

Diverticula

Distensible pouch demonstrable by fluoroscopic observation of meal of thick barium (p 1730).

result in syncope and death. At times this maneuver is successful in stopping a *paroxysmal cardiac irregularity* (p. 873).

The *recurrent laryngeal nerves* lie internal to the common carotid arteries and in close proximity to the thyroid gland hence their liability to injury during *thyroidectomy*. The left recurrent nerve arises from the vagus in front of the aortic arch. It winds around the aorta in close proximity to the left auricle. Dilatation of the aortic arch or the auricle (as in *mitral stenosis*) may cause paralysis of the nerve.

The carotid pulsations are palpated in the sheath. Deep pressure over the carotid bulb may cause syncope or even death in susceptible indi-

DIFFERENTIAL DIAGNOSIS OF

Disturbances of the Salivary Glands

With the exception of mumps abnormalities of the salivary glands are seen with great rarity. The less frequent abnormalities unfortunately may be of great severity as in the instance of neoplasms and chronic infections.

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|-----------------------|--|
| Suppurative parotitis | Following operation and in the course of prolonged illness (p. 1708). Try intensive treatment with antibiotics and radiation. |
| Epidemic parotitis | Mumps with history of exposure (p. 480). Be on alert for complications in testes or ovaries. |
| Mikulicz's disease | May be associated with blood dyscrasia. Check hemogram and biopsy (p. 1709). |
| Sarcoidosis | With associated involvement of uveal tract. Get tuberculin reaction and biopsy (p. 3271). |
| Neoplasms | Mixed tumors and carcinomas of parotid (p. 1714). Get biopsy. |
| Calculous | Of Stensen's or Wharton's ducts. Note acute and chronic swellings. Look for radiopaque shadows (p. 1711). Refer to dental surgeon. |
| Toxic | With iodism. |

viduals. Through the sheath direct pressure is applied to the vagi. Pressure on the left vagus may cause slowing of the pulse rate.

EXTERNAL JUGULAR VEIN

Superficial to the sternomastoid muscle runs the external jugular vein which receives the blood from the scalp and face and terminates in the subclavian or internal jugular vein. In congestive failure the external jugular vein becomes distended. The study of its pulsations may yield important clinical observations (p. 3579).

In infancy *venipuncture* and/or *intravenous infusions* (p. 3775) may be performed in an emergency in the external or internal jugular veins.

DIFFERENTIAL DIAGNOSIS OF

Vascular Disturbances in the Neck

The vascular structures of the upper extremities and head converge in the neck which contains the jugular veins and the innominate subclavian and carotid arteries. Prior to the modern era of electrocardiography considerable attention was directed to tracings which depicted auricular and ventricular activity in carotid artery and jugular vein. While these burdensome examinations are no longer practiced direct observation of the vascular structures may yield valuable information.

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

Throbbing of carotid artery

Neurocirculatory asthenia hyperthyroidism heart block aortic insufficiency hypertension, arteriosclerosis aortic aneurysm, patent ductus arteriosus and arteriovenous aneurysms. Note bradycardia of heart block (p. 879). Tachycardia and elevation and B.M.R. in hyperthyroidism (p. 1197). Elevation of systolic and diastolic pressures in hypertension (p. 910). Increased pulse pressure in aortic insufficiency and hyperthyroidism. Diastolic murmur in aortic insufficiency (p. 973). Machinery murmur in patent ductus arteriosus (p. 957). Get x rays of chest and Ecg.

Inequality of carotids

Aneurysm of aorta or innominate artery (p. 796). Get chest x rays and serologic findings. With pulsation in tricuspid insufficiency (p. 970). With backward failure heart block aortic aneurysm acute and chronic pericarditis cardiac tamponade emphysema and bronchial asthma. Note dyspnea orthopnea cyanosis and edema of backward failure (p. 941). Prolonged expiration in emphysema (p. 2056). Sibilant and sonorous rales in asthma (p. 2101). Distant heart sounds in cardiac tamponade (p. 872). Confirm physical findings with x rays of chest and Ecg.

Collapse of jugular vein

Adhesive pericarditis (p. 1010). Note fixation of apex. Confirm by x ray.

Episternal pulsation

Aortitis and aortic aneurysm (p. 1026). Check chest x ray and serology.

Supraclavicular pulsation

With protrusion of subclavian artery over cervical rib (p. 2953). Aneurysms of subclavian or innominate artery (p. 1026). Check x rays of cervical spine and chest.

Local cyanosis

Superior caval obstruction due to substernal thyroid mediastinal tumor or aortic aneurysm. Check chest x ray.

Thyroid apoplexy

Particularly with longstanding colloid goiter. Note sudden appearance and gradual recession (p. 1208).

widens into the *carotid sinus* which is richly supplied with autonomic nerve fibers. Pressure on the sinus produces a vasovagal reflex and may

The *submaxillary glands* drain into the floor of the mouth through Wharton's ducts which open at the side of the frenum. The *sublingual glands* are drained on each side by a series of eight to twenty ducts which open along a line running laterally from the frenulum.

CERVICAL LYMPH NODES

There are some ninety lymph nodes on each side of the neck. These drain the numerous foci of infection of the nose and throat. The main groups of lymph nodes are

1 The *superficial chain* from the lower pole of the parotid gland along the external jugular vein

2 The *submental group* composed of three small nodes lying in the submental space between the two anterior digastric muscles

3 The *submaxillary group* of three nodes closely applied to the submaxillary gland and draining the lips, the anterior face and tongue

4 The *posterior auricular group* lying on the mastoid insertion of the sternomastoid muscle and draining the posterior scalp

5 The *deep cervical chain* following the internal jugular vein from the jugular fossa to the clavicle. This chain may be subdivided into (a) a *carotid packet* at the carotid bifurcation (b) an *omohyoid packet* just below this (c) a *posterior digastric group* lying beneath this muscle and (d) a *supraclavicular group* around the junction of the external and internal jugular veins

THE CERVICAL FASCIAS AND ROUTES OF SPREAD OF INFECTION IN THE NECK

The Fascial Layers—There are superficial and deep fascias in the neck. (1) The *superficial fascia* is thin and of minor surgical importance. Abscesses forming beneath it point through the skin. (2) The *deep cervical fascia* forms a complete envelope for the neck and sends branches between all its various structures. The deep fascia is subdivided into three chief layers: (a) the *superficial layer* of the deep fascia completely enveloping the neck; (b) the *prevertebral layer* lying in front of the spinal column; and (c) the *pretracheal layer* lying in front of the trachea.

The *superficial layer* of the deep fascia encases the trapezius and sternomastoid muscles. The *prevertebral layer* is attached to the base of the skull and passes inferiorly into the posterior mediastinum. In its inferolateral portion it forms a sheath which passes into the axilla. The *pretracheal fascia* is carried posteriorly to form the *buccopharyngeal fascia* behind the pharynx and esophagus. It is attached to the hyoid bone above and passes inferiorly to the arch of the aorta where it is continuous with the pericardium. The *buccopharyngeal fascia* continues inferiorly into the posterior mediastinum. The *carotid sheaths* are formed by the fusion laterally of the three layers of the deep cervical fascia under the sternomastoid muscles. The carotid sheath follows the great vessels and nerves into the chest and is continuous with the subclavian sheath.

The Fascial Compartments and the Paths of Suppuration—1 The space between the pretracheal and prevertebral fascias contains the trachea, esophagus, thyroid, carotid sheath and brachial plexus. Suppuration here is rare but extremely menacing.

THE SALIVARY GLANDS

The parotid, submaxillary and sublingual glands, which function to produce salivary secretion, are easily palpated. The parotid glands drain

DIFFERENTIAL DIAGNOSIS OF

Cervical Lymphadenopathy

Enlargements of the cervical lymph nodes may be local manifestations of systemic disorders or may represent satellite involvements from inflammatory or neoplastic disturbances of oropharynx, larynx, mediastinum or stomach.

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|--|--|
| Dental infections | Periodontitis, dental caries, dental abscess, stomatitis and gingivitis. Supplement visual findings with set of dental x rays. Refer patient to dentist. |
| Inflammations of oropharynx | Glossitis, tonsillitis, peritonsillar or retropharyngeal abscess, trench mouth or Vincent's angina. In presence of local exudate get smears for bacterial stains, particularly seeking <i>C. diphtheriae</i> and <i>fusci</i> <i>spirochetes</i> . Culture for identification of predominant organism. Initiate antibiotic treatment. |
| Dermatoses | Pediculosis capitis, Pyoderms of face or neck. |
| Systemic infections | Infectious mononucleosis, measles, rubella, scarlet fever, diphtheria, tuberculosis or syphilis. Atypical monocytes in blood smear, positive heterophile reaction and false positive Wassermann tests in mononucleosis (p. 466). Persistent positive serology in syphilis (p. 336). Morbilliform eruption in measles, rubella and infectious mononucleosis (p. 412). Scarlatiniform rash in scarlet fever and infectious mononucleosis (p. 406). Tonsillar or pharyngeal exudate containing organism in diphtheria (p. 302). Positive patch test in tuberculosis (p. 252). |
| Diseases of blood and blood forming organs | Acute and chronic leukemia (p. 1100). Lymphosarcoma (p. 1137). Sarcoidosis (p. 3211). Hodgkin's disease (p. 1138). Follicular lymphoblastoma (p. 1137). Diagnostic hemogram in the leukemias (p. 1100). Pathognomonic cells from sternal biopsy in leukemia. Hodgkin's disease and follicular lymphoblastoma (p. 1035). In doubt, remove node for histologic study. |
| Neoplasms | Metastatic malignancy from skin, tongue, gums, nasopharynx, tonsils, larynx, oropharynx and stomach. Get biopsy for definitive findings. Gastric neoplasm often associated with supraclavicular sentinel node of Virchow (p. 1816). |

into the oral cavity through *Stensen's ducts* which open opposite the second upper molar tooth.

| | |
|------------------------|---|
| Oropharyngeal | Tonsillitis peritonsillar and retropharyngeal abscess foreign body in airway sphenoiditis laryngitis and edema of larynx. Supplement inspection with transillumination of sinuses and indirect laryngoscopy Refer to specialist particularly with evidences of obstructive respiration Start intensive antibiotic therapy and prepare for surgery |
| thyroidal | Particularly with acute tumors resulting from thyroiditis strumitis and thyroid apoplexy Prepare for immediate surgery with obstruction to respiration |
| Lymphatic | Acute varieties of cervical adenitis and Ludwig's angina (p 1708) Try intensive treatment with antibiotics Prepare for surgery if airway is obstructed |
| Pleuropulmonary | Particularly with diaphragmatic pleurisy and basal pneumonia or pneumonitis (p 2185) Confirm physical signs with chest x ray |
| Renal | As result of fulminating pyelitis or pyelonephritis (p 2353) Examine urine Refer to specialist for ureteral catheterization and radiographic studies |
| Circulatory | Angina pectoris coronary insufficiency and coronary occlusion Note effect of nitroglycerin (p 3692) Get Ecg |
| Biliary | With inflammation and colic involving gall bladder Note signs in right upper quadrant Look for jaundice and radiopaque shadows (p 1951) |
| Vascular | Thrombosis of jugular vein (p 2157) In association with subarachnoid or intracerebral hemorrhage Get spinal fluid for evidences of active bleeding or xanthochromia (p 3735) |
| Generalized infections | Particularly typhoid (p 225) and rheumatic fevers (p 186) Get blood culture and serology Note therapeutic response to salicylate (p 198) Following infection of penetrating wound with Cl tetani (p 294) |

2 Infection in the *carotid sheath* dissects toward the superior mediastinum and subclavian sheath It may burrow deep to produce *edema of the larynx* (p 2101)

3 Pus between the pretracheal and prevertebral layers follows the trachea and esophagus into the posterior mediastinum Pressure from suppurative or from growths is extremely dangerous in this compartment

4 Pus posterior to the prevertebral fascia (cervical Pott's disease) may follow the scalenus muscle and brachial plexus and appear in the axilla

5 Suppuration in the space between the superficial layer of the deep fascia and the pretracheal layer is frequent points forward and is least dangerous

DIFFERENTIAL DIAGNOSIS OF

Pain and Stiffness of the Neck, Including Opisthotonos

The symptoms of painful stiff neck with opisthotonos may arise from a variety of local disturbances as well as abnormalities involving remote areas. The accessibility of local structures to examination somewhat simplifies diagnostic problems but in the absence of obvious findings extensive investigation may be necessitated.

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|---------------------------|---|
| Subcutaneous tissue | Cellulitis phlegmon and carbuncle. Combine intensive treatment with antibiotics and surgery. |
| Muscular | Following exposure to wind and cold. Tort. collis (wry neck). Myogelosis with painful in durations usually in posterior occipital region. Relieved by massage. Hematoma of sternomastoid particularly in the newborn. Scalenus anticus syndrome with radiation of pain to shoulder and arm and obliteration of radial pulse. Look for cervical rib. Consider division of muscle. Spasm in strychnine poisoning. Increased tonus in tetanus. |
| Articular | Osteo-arthritis of cervical spine in obese middle aged patients with low B.M.R. (p 2855). Rheumatoid arthritis of spine (Marie-Strumpell) in younger patients with vasomotor instability and rapid sedimentation time (p 3707). Subluxation of posterior articular facet causing acute crick in neck. Demonstrate by lateral oblique x rays. Relieved by traction and manipulation. |
| Osseous | Fractures demonstrable by anteroposterior and lateral x rays. Osteomyelitis of skull and of sphenoid-ethmoid. Demonstrable by x ray. Tuberculosis of spine (Pott's disease) (p 2942). Mastoiditis particularly with subcutaneous abscess (p 2146). |
| Meningeal Irritation | Particularly with suppurative sphenoid-ethmoiditis. Get intranasal and x ray evidences of accessory nasal sinus infection (p 2125). Summon specialist for surgery. |
| Suppurative meningitis | Meningococcal pneumococcal influenza streptococcal and staphylococcal invasions either systemic otogenic or rhinogenic. Obtain spinal fluid and identify pathogen (p 3782). Look for primary focus in nose or ears. Institute intensive antibiotic therapy. |
| Nonsuppurative meningitis | With clear cerebrospinal fluid as in tuberculosis the virus encephalitis poliomyelitis and lymphocytic choriomeningitis (p 442). If coagulum is present make careful examination for tubercle bacilli. |

of the breasts See *Differential Diagnosis of Clinical Disturbances of Breasts and Nipples* (p 2578)

DIFFERENTIAL DIAGNOSIS OF

Visible Abnormalities of the Chest Wall

Other than disturbances of the breast and nipple elsewhere discussed (p 2533) Inspection of the chest wall may reveal abnormalities of great local or systemic significance

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|------------------------|---|
| Congenital | |
| Sprengel's deformity | Congenital elevation of scapula Usually unilateral (p 2823) |
| Mechanical | |
| Barrel chest | See p 3488 |
| Phthisical chest | See p 3489 |
| Malunion | Particularly fractures of clavicles and ribs Get x rays |
| Scars | Following burns and surgical procedures such as thoracoplasty |
| Asymmetries | Due to kyphosis and scoliosis (p 3059) Muscle atrophy secondary to poliomyelitis hemiplegia or dystrophies |
| Neoplastic | |
| Sebaceous cysts | Usually posterior and freely movable May reach large size (p 3935) Excise if possible |
| Multiple myeloma | May involve ribs Bence Jones proteose in urine Myeloma cells in sternal biopsy Areas of destruction by x ray (p 1126) |
| Metastatic carcinoma | Particularly of ribs Areas of destruction revealed by x rays |
| Metabolic | |
| Rickets | Rosary at costochondral junctions Harrison's grooves Pigeon or chicken breast Funnel or cobbler's breast Therapeutic response to vitamin D (p 2850) |
| Enlargement of xiphoid | Causes epigastric mass suggesting gastric neoplasm |
| Inflammatory | |
| Syphilis | Gumma may involve ribs or sternum Therapeutic response to iodide |
| Empyema necessitatis | Pointing of pleural suppurative process Aspirate pus Confirm findings by x ray (p 2219) Refer to surgeon |
| Tuberculosis | Cold abscess often secondary to osteomyelitis of spine Obtain biopsy of wall X ray chest (p 2199) |
| Dermatoses | See p 3097 |

Inspection—The breasts are inspected with the patient recumbent seated and finally on her knees and elbows The breasts may vary from the small pointed structures of the adolescent child to the sagging pen

CHAPTER 159

THE PHYSICAL EXAMINATION OF THE THORAX

EXAMINATION of the thorax is simplified by the separate consideration of (1) the *thoracic wall* (2) the *thoracic cage* and (3) the *thoracic cavity* and its contents

THE THORACIC WALL

ANATOMIC REVIEW

The clinical investigation of the thoracic wall includes the direct examination of its inherent structures and the delineation of the projections of the more important contents of the thoracic cavity

Clavicles—Above and anteriorly the surface of the thorax is marked by the *clavicles* which may be palpated throughout their entire length. The *sternoclavicular articulations* join clavicles and *sternum* the anterior surface of which is readily felt

Suprasternal Notch—Through the suprasternal notch the examining finger may palpate substernal extensions of the thyroid gland and occasionally the contents of the anterior mediastinum especially the pulsating arch of the aorta. This pulsation is marked in cases of *aneurysm* (p 1026) and is absent in *aortic stenosis* (p 971)

Sternal Angle—The junction of the *manubrium* and the body of the *sternum* produces a prominence (the sternal angle) which marks the level of the second rib anteriorly. It is in the region of the angle that the sternum is usually aspirated to obtain marrow (p 1035)

Xiphoid Process—The sternum ends below in the *infra-sternal notch*. Below the notch the *xiphoid process* can be felt. At times this forms a prominent tumorlike projection. Patients often seek consultation believing that this lump represents some type of intra-abdominal neoplasm.

Free Borders of Ribs—Laterally the free borders of the ribs are palpated for points of tenderness. *Subluxation* of the ends of the free ribs may cause misleading symptomatology simulating visceral disease

Muscles and Lymph Nodes—On either side of the sternum there is the prominence of the underlying *pectoral muscles*. The *subpectoral region* contains a group of lymph nodes draining the upper extremities. These may become involved in suppurative inflammation (*subpectoral abscess*) and give rise to considerable diagnostic and surgical difficulty

Vessels—The *internal mammary vessels* run parallel to the lateral borders of the sternum. They emerge below the ribs as the *superior epigastric vessels* communicating with the *inferior epigastric* branches of the external iliacs (see Portal Obstruction). The internal mammary arteries may become markedly dilated and tortuous to form an effective collateral circulation in cases of *coarctation of the aorta*. Similarly in instances of *obstruction to the superior vena cava* or *portal vein* the superficial chest and abdominal veins become engorged and prominent. When present to a mild degree this may be clearly depicted by *infra-red* photography

The Breast—Overlying the pectoral region is the mammary tissue. The male breast consists of glandular tissue and nipple as in the female. The structure is rudimentary but may develop inflammatory and neoplastic changes similar to those of the female breast

THE FEMALE BREAST

A meticulous survey of the female breasts is an integral part of the routine physical examination. Through this discipline the earliest diagnosis of *mammary cancer* is established. There can be no greater advance in the prophylaxis of malignancy than insistence upon routine examination

of the breasts See *Differential Diagnosis of Clinical Disturbances of Breasts and Nipples* (p 2578)

DIFFERENTIAL DIAGNOSIS OF

Visible Abnormalities of the Chest Wall

Other than disturbances of the breast and nipple elsewhere discussed (p 2533) inspection of the chest wall may reveal abnormalities of great local or systemic significance

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|------------------------|---|
| Congenital | |
| Sprengel's deformity | Congenital elevation of scapula Usually unilateral (p 2823) |
| Mechanical | |
| Barrel chest | See p 3488 |
| Phthical chest | See p 3489 |
| Malunion | Particularly fractures of clavicles and ribs Get x rays |
| Scars | Following burns and surgical procedures such as thoracoplasty |
| Asymmetries | Due to kyphosis and scoliosis (p 3059) Muscle atrophy secondary to poliomyelitis hemiplegia or dystrophies |
| Neoplastic | |
| Sebaceous cysts | Usually posterior and freely movable May reach large size (p 3935) Excise if possible |
| Multiple myeloma | May involve ribs Bence Jones proteose in urine Myeloma cells in sternal biopsy Areas of destruction by x ray (p 1126) |
| Metastatic carcinoma | Particularly of ribs Areas of destruction revealed by x rays |
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| Dermatoses | See p 3097 |

Inspection—The breasts are inspected with the patient recumbent seated and finally on her knees and elbows The breasts may vary from the small pointed structures of the adolescent child to the sagging pen-

dulous breasts of the obese multipara Asymmetry or abnormality of contour requires careful investigation

The *nipple* is closely scrutinized There should be no discharge other than in the lactating phase Serous or sanguineous secretion is regarded with suspicion

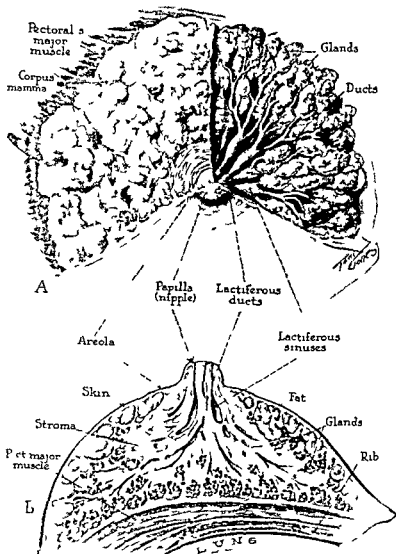


Fig. 1023—A Dissection of lactating breast B Relation of breast to chest wall

The *areola* (Fig 1023) of the nipple frequently contains small superficial *adenomas*. They may be ignored unless they are inflamed or surrounded by induration. An increase in the brown pigmentation of the areola occurs during pregnancy and is present during lactation. In the vicinity of the areola it is not uncommon to find *bristly hairs*.

Palpation—To palpate the breasts the physician sits behind the patient and encircles her body with his arms. With his left hand he cups and supports the left breast and palpates with the fingertips of his right hand. The positions of the hands are reversed for the examination of the right breast.

When the examination has been completed from behind the examiner faces the patient and with thumb and forefinger gently pinches small areas of each breast. Occasionally a very small abnormality of the mammary tissue is felt in this way.

Later when the patient is taken into the dark room (p 3588) palpatory transillumination is focused on the suspicious areas. In this way tumors and cysts may be delineated in silhouette and blood vessels may be seen coursing to inflammations or neoplasms.

Examination of Tissue—Suspicious areas in the breast particularly if they evidence growth require *puncture* or *biopsy* to obtain material for *histological examination* (p 3935).

ACCESSORY MAMMAE

Accessory nipples may occur along the course of the *mammary ridge*. These are subject to the same disturbances as the normally situated tissue.

AXILLARY LYMPH NODES

Since breast drainage travels towards the axillary nodes this region is examined by palpation as an integral part of the examination. The presence of a unilateral axillary lymphadenopathy without adequate explanation directs the practitioner to review again the examination of the breast.

THE THORACIC CAGE

The thoracic cage is a bony framework formed by the vertebrae posteriorly, the ribs laterally and the sternum anteriorly. It is normally symmetrical but congenital and acquired deformities are not uncommon.

SHAPE AND TYPES

The thoracic cage is nearly cylindrical in the infant and child, its anteroposterior diameter approximating the lateral. In adult life the chest tends to flatten in the anteroposterior plane. The conformation of the cage may be of the linear, stocky or normal type.

In the Linear Type—In individuals with the linear type of thoracic cage (p 3488) the chest is long, thin and narrow, the free ribs extend almost to the pelvic crest and the costal angle is acute. The heart is long and narrow and the diaphragm and liver are situated low. The examiner anticipates a diminished transverse cardiac diameter and hypotension. It is not unusual for the liver edge to be normally palpable. This type of chest has been referred to as *phthisical* because of the frequency with which it is observed in tuberculosis.

In the Stocky Type—In the *stocky*, *apoplectic* or *barrel chested* type of individual the thoracic cage resembles that of the infant. The anteroposterior diameter approaches that of the lateral, the free ribs are well

above the pelvic crest, and the costal angle is oblique. The heart is wide and the diaphragm is situated high in the thorax. As a result radiographs reveal a surprisingly limited respiratory area, particularly in women with pendulous breasts. The exaggeration of barrel chest occurs in *emphysema* (p. 2056).

DIFFERENTIAL DIAGNOSIS OF

Axillary Lymphadenopathy

The important axillary lesions are referable to involvements of the lymph nodes which drain extremities and breasts.

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

Local Infection or Inflammation

Contact Dermatitis

From depilatories and androgens

Axillary Sweat Gland Abscess

Producing multiple and recurrent lesions (p. 3253). Try local and systemic treatment with penicillin.

Lymphadenitis

Usually from finger or hand infection. With vaccinia. Primary lesions of syphilis and tertiary syphilis involving fingers. Get darkfield microscopy and bacteriologic studies before institution of antibiotic therapy (p. 45).

Intertrigo

From sweating and maceration of skin, particularly in obese (p. 3161).

Systemic Infection

Infectious Mononucleosis

Generalized lymphadenopathy. Atypical monocytes in blood and positive heterophile reaction. May have false positive Wassermann (p. 466).

Syphilis

In secondary stage with positive serology. Examine aspirate by darkfield microscopy (p. 45).

Tuberculosis

Chronic lymphadenitis with caseation. Get patch test, biopsy and chest x-ray (p. 556).

Leukemia

With atypical cells in peripheral blood or bone marrow (p. 1100).

Hodgkin's Disease

Chronic granuloma. Usually associated with sustained fever and splenomegaly. Diagnose by biopsy (p. 1138).

Neoplasm

Cystic lymphangioma (p. 3203). Metastatic especially from malignancy of breast (p. 2581).

In the Normal Type—The normal chest lies midway between the extremes of the linear and stocky types.

ZONES OF THE THORAX

In thoracic investigation several arbitrary lines and areas are conventionally recognized. These terms are self-explanatory. They include the

midsternal line anteriorly the midline of the back posteriorly the *para sternal sternal mammary anterior mid and posterior axillary lines*

Lesions are described as occurring in various areas such as the *supra claviclar infraclavicular mammary axillary suprascapular scapular interscapular and infrascapular regions*

REFLECTIONS OF THORACIC VISCERA ON THORACIC WALLS

Since the thoracic viscera cannot be observed or palpated through the bony thoracic cage their anatomical structures and pathological conditions are determined indirectly by percussion auscultation and roentgenography. It is therefore important to learn the projections and reflections of the deeper viscera. This becomes of practical significance in the exact localization of a *lung abscess* (p 2214) preparatory to determining the site for operative procedure since a poorly placed incision exposes the free pleural cavity and favors extension of infection.

ANATOMIC REVIEW

The Lobes of the Lung.—On the left side the lung is divided into an upper and lower lobe. The *te lobar fissure* may be projected externally on the thorax by a line starting at the spine of the fourth thoracic vertebra crossing the fourth and fifth ribs in the axilla and terminating anteriorly and medially just above the sixth rib. Thus the *left upper lobe* underlies virtually the entire anterior surface of the left chest. Posteriorly its area is limited to the region above the fourth rib.

On the right side the lung is divided into three lobes. The lower border of the upper lobe extends from the fourth thoracic vertebra posteriorly to the fourth rib anteriorly. The projection of the *middle lobe* is triangular in shape. Its apex is in the mid axillary line just below the fifth rib the base in the midsternal line from the fourth to the seventh ribs. The upper border of the *middle lobe* on the lower border of the upper lobe posteriorly and on the mandibula. From this point on it impinges upon the lower border of the middle lobe. Its projection extends from the level of the fourth thoracic vertebra posteriorly crossing the fourth fifth and sixth ribs to terminate anteriorly just above the level of the seventh rib.

The anterior thorax on the right side is made up almost entirely of the upper and middle lobes. The axillary portion reflects all three lobes. Posteriorly except for the area underlying the first three ribs the entire surface overlies the lower lobe.

Projection of the Heart.—Position of the *cardiac border* places them farther most part to the left of the sternum. The actual borders are determined by percussion.

Along the right border of the sternum there is a *lunar area* of relative dullness that corresponds to the *right auricle*. The *left border* of the heart is delineated from the second to the fifth or sixth left intercostal space. This border is composed almost exclusively of *left ventricle*. It extends approximately 2 to 4 cm to the left of the mid sternal line in the third interspace and reaches a distance of 7 to 9 cm to the left of the midsternal line in the fifth or sixth left interspace. This left border is covered by the lingular portion of the left upper lobe hence dullness relative and the apex impulse is palpated through intervening pulmonary tissue.

The overlapping of the heart by pulmonary tissue is variable. In almost all instances there is a triangular area of *ab lute cardiac flatness* corresponding to that portion of the pericardium which is directly beneath the thoracic cage and to the left of the sternum.

Projection of Other Structures.—Besides the heart and lung the upper border of the liver may be projected on the chest. All the upper border of liver dullness begins at the lower border of the fourth rib and only (Figs 1013) a line extends to the level of the sixth or seventh rib. Throughout this area the dome of the liver is separated from the chest wall by intervening lung and diaphragm. From the third to the free costal border liver flatness may be percussed provided the site is not obscured by the tympany of distended gut.

An area of *supracardiac dullness* exists beneath the manubrium of the sternum. It represents the underlying great vessels.

DIFFERENTIAL DIAGNOSIS OF

***Abnormal Movements and Pulsations
of the Thoracic Cage***

Inspection of the thoracic cage may reveal peculiarities in movement, abnormal pulsations or retractions. Any of these phenomena requires intensive supplementary examination, including x rays for the detection of the causative mechanism.

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|----------------------------------|---|
| Bulging of intercostal spaces | With increased resonance or tympany (emphysema, tension pneumothorax or bronchial asthma). With dullness to flatness (pleural effusion, pericardial effusion or empyema thoracis). Obtain x ray. Perform diagnostic thoracentesis (p 2222) or pericardiocentesis (p 852). |
| Retraction of intercostal spaces | Acute atelectasis (p 2054) or fibrosis of lungs (p 2199). X ray chest. |
| Increased chest expansion | In athletes. Acidosis and hypercapnia (p 721). Bradypnea associated with morphine poisoning (p 2014). |
| Decreased chest expansion | With bulging chest (emphysema, bronchial asthma, hydrothorax and pneumothorax). With chest retraction (fibrosis and atelectasis of lungs, chronic adhesive pleurisy, acute pleurisy and respiratory failure). X ray chest. Perform diagnostic thoracentesis (p 2222). |
| Asymmetrical expansion | Unilateral pleuropulmonary disease. Unilateral muscle weakness or paralysis (poliomyelitis, hemiplegia). Unilateral paralysis of diaphragm especially following phrenic division or avulsion (p 2039). |
| Precordial bulge | Pericardial effusion, congenital cardiac disorders or cardiac hypertrophy and dilatation. Obtain x ray and Ecg. Perform diagnostic pericardiocentesis (p 852). |
| Enlargement of superficial veins | Obstruction of superior vena cava or innominate vein (substernal thyroid and mediastinal tumor, lymphadenopathy or infection). Aneurysm of aorta (p 798). Portal obstruction, as in cirrhosis of the liver (p 1969). Thrombosis of portal vein or obstruction of inferior vena cava (p 1960). |
| Abnormal pulsation | Episternal (aortic dilatation or aneurysm). In upper left interspaces (dilatation of pulmonary conus or aortic aneurysm). X ray chest. |
| Abnormal systolic retraction | Chronic adhesive pericarditis (Broadbent phenomenon) (p 1011). |
| Increased apex impulse | Following excitement or effort. In the puffed chest. With hypertension, hyperthyroidism, mitral stenosis and aortic insufficiency. Note cardiac murmurs (p 972) in valvular defects. Increased B.M.R. in hyperthyroidism. |
| Decreased apex impulse | In obesity. With emphysema, myelomalacia, aortic stenosis, pericardial effusion and backward failure. Get x ray of chest and Ecg (p 803). |

| | |
|--------------------------------|--|
| Shift to left of apex impulse | Hypertrophy of left ventricle (p 867) Right pleural effusion (p 2219) Pneumothorax or left atelectasis (p 2054) Get x ray of chest and Ecg (p 803) Diagnostic thoracentesis (p 2222) |
| Shift to right of apex impulse | Left pleural effusion or pneumothorax Right atelectasis Get x ray of chest Diagnostic thoracentesis (p 2222) |
| Fixation of apex impulse | Chronic adhesive pericarditis (p 1010) |

THE MOVEMENTS OF THE THORACIC CAGE

In normal inspiration the ribs move outward and upward elevating and expanding the chest. With expiration the ribs move downward and

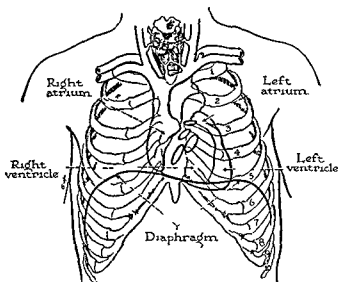


Fig 1024—Position of heart in chest

inward flattening and shortening the thorax (Fig 1012). The respiratory movements may be observed or palpated. Unilateral changes are always indicative of abnormality.

Movements of the *diaphragm* are suggested by the behavior of the costal flare (Fig 1013). With depression of the diaphragm during inspiration the flare is outward, the costal angle becoming more obtuse. During the elevation of the diaphragm in expiration the flare is inward, the angle becoming more acute. When the leaves of the diaphragm function normally the behavior of the costal arches should be equal.

NORMAL CARDIAC PULSATIONS

In the normal individual of thin or average size the normal cardiac pulsation is observed in the fourth, fifth, or sixth interspace, 8 to 10 cm

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Shift to left of apex impulse

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Shift to right of apex impulse

Left pleural effusion or pneumothorax Right atelectasis Get x ray of chest Diagnostic thoracentesis (p 2222)

Fixation of apex impulse

Chronic adhesive pericarditis (p 1010)

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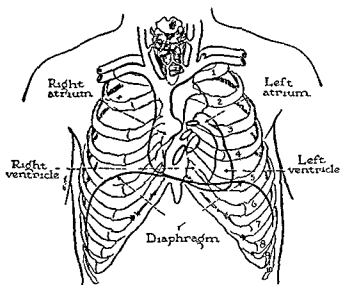


Fig 1024.—Position of heart in chest

inward flattening and shortening the thorax (Fig 1012) The respiratory movements may be observed or palpated Unilateral changes are always indicative of abnormality

Movements of the *diaphragm* are suggested by the behavior of the costal flare (Fig 1013) With depression of the diaphragm during inspiration the flare is outward the costal angle becoming more obtuse During the elevation of the diaphragm in expiration the flare is inward the angle becoming more acute When the leaves of the diaphragm function normally the behavior of the costal arches should be equal

NORMAL CARDIAC PULSATIONS

In the normal individual of thin or average size the normal cardiac pulsation is observed in the fourth fifth or sixth interspace 8 to 10 cm

from the midsternal line. The pulsation should consist of a forward thrust synchronous with systole. In heavy individuals with barrel chests (p 3490) or in females with pendulous breasts the normal impulse may be neither visible nor palpable.

The location of the impulse is important as a check on cardiac percussion since the left border of the heart usually extends no more than 2 cm lateral to the position of the thrust. Normally the apex impulse alters with change of position. The shift should be no less than 2 to 4 cm. Fixation of the impulse suggests adhesive pericarditis (p 1010).

THE POSTERIOR ASPECT OF THE THORACIC CAGE AND THE AXILLA

The structures of the posterior aspect of the thoracic cage and the axilla are discussed under considerations of the *back* (p 3569) and the *upper extremity* (p 3573).

THE THORACIC CAVITY

ANATOMIC REVIEW

The cavity of the thorax contains the *heart* and *great vessels*, the *trachea*, *bronchi* and *lungs*, the *esophagus*, the *thoracic duct*, the *vagi*, *phrenic*, *left recurrent laryngeal* and *splanchnic nerves* and the *costals* and *lymphatics* concerned with the local structures.

The thorax is divided into two pleural cavities by the *mediastinum* which includes the heart, the great vessels, the trachea, the main bronchi, the esophagus, the thoracic duct and nerves.

The Pleura—Each pleural cavity is filled almost completely by the *lung*. The cavity is lined by the glistening *parietal pleura* which is continuous with the *visceral pleura* at the hilus of each lung. The pleural surfaces are in contact over the upper portions of the lungs but enclose a free space in the angle between the thorax and diaphragm (*costophrenic sinus*).

Trachea and Bronchi—The *trachea* is a wide tube beginning at the lower border of the cricoid cartilage and ending at the bifurcation at the superior border of the fifth thoracic vertebra. It varies in length from 4 to 5 inches. It is kept patent by dense cartilaginous rings which are incomplete posteriorly so that a flat surface presents itself to the membranous esophagus posteriorly. It is usually fixed above and stretches with motion of the head and neck. It follows the curves of the spine and hence recedes from the skin as it descends into the thorax.

In the neck the trachea is surrounded by the isthmus and lobes of the thyroid gland, the carotid sheath laterally and the esophagus posteriorly. The recurrent laryngeal nerves are on either side in the groove between esophagus and trachea. In the midline it contains some fascia which separates it from the sternohyoid and thyrohyoid muscles on either side. This *linea alba* is the best surgical approach for tracheotomy. Occasionally a large thyroid ima artery ascends on the anterior surface. The innominate artery is situated between the trachea and the manubrium sternum.

In the chest the trachea is in relation to the arch of the aorta and its branches in front and to the left on the right to the vagus nerve and the right apex of the pleura on the left to the subclavian artery and the recurrent laryngeal nerve.

The *right* and *left main bronchi* are formed by the bifurcation of the trachea and run obliquely downward and outward to the periphery of each lung. The *carina* usually lies slightly left of the midline so that the right bronchus is more a direct continuation of the trachea and usually lodges foreign bodies (p 2075). The left main bronchus is longer than the right. At the hilum are bronchial lymph nodes which drain the pulmonary and bronchial structures.

The *right main bronchus* is subdivided into upper, middle and lower lobe branches. On the left there is no middle lobe. Except for this variation the branches of the left upper lobe correspond to the right.

The Lungs—The lungs covered by their plural layers fill up the respective pleural cavities. The *lung apex* projects above the clavicle. Percussion of this portion gives rise to the area of resonance known as the *shoulder strap*. The apices, particularly the right, are sites of election for the early *tubercle* lesion and its resultant overlying pleurisy. (p 219)

so that adhesions are more often encountered than not. The *bases of the lungs* are concave and rest on the leaves of the diaphragm. The outer surfaces are convex and in apposition to the rib. The inner surface is concave to accommodate the structures of the mediastinum.

The *root of the lung* on either side is formed by the bronchus, the corresponding branch of the pulmonary artery and accompanying pulmonary vein, the pulmonary plexuses of lymphatic vessels and nodes bound together by areolar tissue and covered by pleura. The roots of the lungs lie opposite the bodies of the fifth, sixth and seventh thoracic vertebrae between the median line and the vertebral borders of the scapulas. Just above the root the main bronchi join to form the *trachea*, usually at the level of the lower part of the body at the fourth thoracic vertebra. Here are found the *tracheobronchial lymph nodes* which are usually divided into four groups—a right pretracheobronchial group, a left pretracheobronchial group, an intertracheobronchial group and an interbronchial group. These hilar glands are often involved in metastatic lesions, tuberculosis and especially the infectious granulomas such as *Histoplasma disease* (p. 1139). The reflections of the lung borders, fissures and lobes of the thoracic cage are else here described (p. 3597).

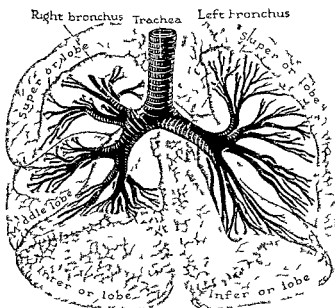


FIG. 10.—The lungs with trachea and bronchi.

Within the lungs the *bronchi* divide usually dichotomously until the finest bronchiole terminates in the atrium from which a number of thin-walled *alveoli* open out. In the terminal air sac the interchange of gases alters the blood which receives oxygen and discharges carbon dioxide. The finer bronchi are encircled by *smooth muscle* under the control of the involuntary nervous system. Spasm of this tissue produces the characteristic symptomatology of *bronchial asthma* (p. 101). The motor innervation of the muscle is via the cholinergic subdivision, inhibitory fibers being derived from the adrenergic portion.

The *lung epithelium* of the alveoli undergoes gradual changes from above downward. In the larger bronchi there is ciliated stratified or pavement variety. In the air cells the lung epithelium is flattened to provide easier interchange between the atmospheric air and the rich capillary plexus that surrounds each alveolus.

The *Epithelium*—See page 1720.

The *Mediastinum*—The lungs are separated by the mediastinum which is divided into anterior, middle, superior and posterior portions.

The *anterior mediastinum* is composed of loops of the lymphatic lymphatics and some smaller branches of the internal mammary artery. The pericardial sac is in contact with the heart.

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tains the arch of the aorta and its three main branches the innominate veins the upper portions of the superior vena cava the esophagus and trachea the thoracic and right lymphatic ducts and the remains of the thymus and some lymph nodes The posterior mediastinum is a continuation downward of the superior mediastinum and it contains the descending aorta the esophagus thoracic duct, azygos veins and the vagi

The middle mediastinum is occupied by the heart and pericardium the ascending aortic arch the lower portion of the superior vena cava the end of the vena azygos major the pulmonary artery and its two main branches and the pulmonary veins and the tracheo-bronchial lymph nodes The upper portions of the phrenic and the pulmonary branches of the vagi also lie in the middle mediastinum The anatomy of the heart and the remaining structures of the vascular system are elsewhere described (p 3545)

Of the contents of the thorax the heart and its great vessels the bronchial tree the lungs and at times the pleural cavity may be investigated by physical examination

CLINICAL EXAMINATION OF CONTENTS OF THORACIC CAVITY

PALPATION

Palpation of the thorax adds confirmatory evidence to the results of inspection The extent and symmetry of the *respiratory movement* may be felt The *apex impulse* is more accurately localized by digital pressure

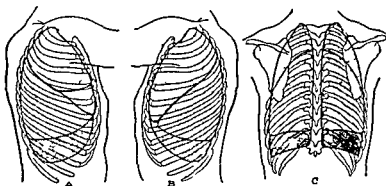


Fig 1026—Thorax showing surface relation of lungs and pleurae The heavily shaded areas represent the full extent of pleural cavities beyond the resting lungs

Under pathological conditions *friction rubs* and *rales* of pulmonary origin *thrills* *coarse murmurs* and *accentuation of sounds* of circulatory origin are felt as well as heard These are better appreciated aurally and are later described (p 3538)

The *apex impulse* of the heart usually is felt in the fifth intercostal space 8 to 10 cm from the midline In the barrel type of chest the apex impulse lies higher and more laterally in a linear type of thorax the apex impulse is situated lower and nearer the midline

After palpating the apex impulse the anterior chest is felt with the flat of the hand for *abnormal pulsations* *shocks* *friction rubs* and *thrills* If present these signs should be carefully localized and interpreted (p 3534)

PERCUSSION AND AUSCULTATION

Following inspection and palpation of the thoracic cage the lungs are examined by percussion and auscultation These two auditory methods complement one another each being dependent upon the physics of sound

Technic of Percussion—Satisfactory percussion is readily learned. Contrary to the general belief unusual sensitivity of the ear is not necessary. The audible variations are well within the range of the average ear and good percussion has been satisfactorily practised by many practitioners who have impairment of hearing. For accurate percussion the practitioner needs only the fingers of his hands. The middle finger of one hand is commonly employed as the *pleximeter* (that which is struck). The index or middle finger of the opposite hand is used as the *plexor* (that which strikes).

Despite the simplicity of percussion certain technical points are to be observed. Good percussion is very much like good putting. It seems absurdly simple yet requires both concentration and precision. The *pleximeter* finger must be placed firmly over the area of investigation. Unsatisfactory percussion usually results from a wobbly contact between the *pleximeter* finger and the chest wall. To prove this the practitioner need only observe the difference in sound produced with equal *plexor* strokes and varying degrees of pressure by the *pleximeter* finger. Although the stroke of the *plexor* finger or fingers is a simple procedure it must be precise. The movement is strictly in the wrist. It is practised by hyperextending the wrist and then permitting the *plexor* fingers to fall almost by gravity directly upon the central portion of the terminal phalanx of the *pleximeter* finger preferably just behind the nail bed. If the blow falls laterally the sound will be marred. A blow on the nail will cause a click. A blow on the second phalanx may be imperfect due to poor contact between this portion of the finger and the chest wall.

For *light percussion* the arc of excursion of the *plexor* finger is but 2 or 3 inches. For *deep percussion* the arc may be increased to from 6 to 8 inches. Instrumental *plexors* and *pleximeters* are unnecessary. The human fingers are equally satisfactory; moreover it is difficult even for the absent minded practitioner to leave home without them.

The Position of the Patient During Percussion—For percussion of the *anterior* and *axillary* portions of the chest the patient should be recumbent with his hands clasped over his head. In this position the borders of the heart are best delineated. Later the patient sits upright and the supraclavicular regions are percussed. For percussion of the *back* the patient sits erect, folds his arms and permits the head to sag. Only in cases of severest illness should the back be percussed with the patient in the lateral recumbent position. Under these circumstances the note in the dependent chest is the more reliable while the findings in the uppermost chest are often completely misleading.

The Percussion Sounds—The basic percussion sound is that of *normal resonance*. This is produced when the underlying pulmonary tissue possesses a normal amount of air within the individual alveoli.

Normal Resonance—The normal resonant note is sounded first in much the same way that an orchestra is given the pitch note. Normal resonance is best elicited in the infraclavicular regions and below the angle of the scapula. At these sites the vesicular sound is not modified by adjacent bronchi. There are no words to describe normal resonance to which the ear must become attuned. Resonance is the normal respiratory sound.

DIFFERENTIAL DIAGNOSIS OF

Abnormalities of Chest Wall Noted on Palpation

Palpatory exploration of the chest wall reveals tenderness, circulatory pulsations and thrills and the vibratory sensations produced by speech. Special consideration has been given elsewhere to abnormalities of breasts and nipples (p 2578).

CLINICAL OBSERVATION CAUSES AND DIAGNOSTIC FEATURES

| | |
|--|--|
| Tenderness of skin | Herpes zoster with eruption (p 435). Lile costal neuralgia with demonstrable sensory disturbances (p 1491). Head zone s due to visceral lesions (p 1474). |
| Tenderness of muscle | Myogelosis and myositis. Relief from massage, local heat and analgesics (p 2864). |
| Tenderness of clavicles, ribs or sternum | Fracture demonstrable by x ray. Osteomyelitis with evidences of inflammation (p 2930). Neoplastic deposits due to primary growths, metastases or multiple myeloma (p 1176). Obtain x rays. Consider biopsy. Examine urine for Bence Jones proteose (p 3573). |
| Tenderness of chest wall | Over areas of acute pleuritis, acute pericarditis or pulmonary abscess. Supplement physical examination with x rays. |
| Tenderness of costochondral junction | Slipping rib with localized tenderness. Relief from procaine injection. |
| Increased fremitus | Normal in linear chest (p 3488). Over area of pulmonary consolidation. Get sputum and x ray. |
| Diminished to absent fremitus | Normal in obesity and with barrel chest (p 3489). With dullness to flatness (pleural effusion, empyema, pulmonary atelectasis and collapse, solid tumors of pleura or lungs, passive congestion or pleural fibrosis). Confirm physical examination with x ray. Consider diagnostic thoracentesis (p 2222). With hyperresonance (emphysema, pneumothorax, bronchial asthma and eventration or herniation of the diaphragm). Supplement physical examination with x ray. |
| Friction rub | Acute fibrinous pleuritis or pericarditis. Note relief from immobilization. |
| Thrills and shocks at apex | Presystolic. In mitral stenosis, aortic insufficiency (Austin Flint) and hyperthyroidism (pseudo thrill). Get x rays for changes in cardiac silhouette. Note increased basal metabolic rate in hyperthyroidism. |
| Thrills and shocks at base | Diastolic with aortic insufficiency (p 972). Systolic with aortic and pulmonary stenosis (p 972). Confirm physical signs with outline of cardiac silhouette. Get Ecg particularly for axis deviation (p 803). |

| | |
|--|---|
| Thrills and shocks in upper left interspaces | Aortic aneurysm patent ductus arteriosus or interventricular septal defects Confirm physical signs with fluoroscopy for cardiac silhouette Get chest x ray and Ecg |
| Continuous thrill | Patent ductus arteriosus (p 937) |
| Whirring sensation | Cysts and abscesses due to amebiasis (p 523) or echinococcus disease (p 2044) Look for eosinophilia in peripheral blood Examine stools for amebae and hooklets Consider diagnostic aspiration. |
| Forcible apex beat | Normal in linear chest (p 3488) With mitral stenosis aortic insufficiency hypertension, hyperthyroidism and neurocirculatory asthma Confirm physical signs with outline of cardiac silhouette and Ecg Note increased basal metabolic rate in hyperthyroidism. |
| Feeble apex beat | Normal with barrel chest (p 3489) With emphysema pleural effusion, pericardial effusion, myelomalacia, backward failure coronary occlusion and aortic stenosis Confirm physical signs with chest x ray and Ecg Consider diagnostic aspiration |
| Displacement of apex beat | Cardiac dilatation and hypertrophy (p 867) Contralateral with pleural effusion and pneumothorax (p 2035) Ipsilateral with massive collapse of lung and fibrosis Confirm physical signs with chest x ray Consider diagnostic thoracentesis (p 2222) |

heard throughout the chest except where modifications are produced by the presence of the solid viscera or the larger bronchi

Normal modifications of resonance are encountered over the liver the heart and the left lower ribs anteriorly in the median line anteriorly and posteriorly the sound being altered by the presence of spine trachea and larger bronchi and in the supraclavicular region where the sound is changed by the proximity of the trachea

Modifications of Resonance—The main qualitative modifications of resonance depend upon the amount of air in the alveoli The range of sound passes from resonance to hyper resonance and tympany in the one direction dependent upon progressively increased aeration of the alveoli In the other direction the changes vary from dullness to flatness with lesser aeration of the terminal radicles

1 **Hyper resonance**—With a simple increase in the content of the alveolar air as in *emphysema* the resonant note increases in sonority and the sound is described as hyper resonant

2 **Tympany**—When in addition to an increase in the air content of the alveoli a tense or distended wall or membrane is set into vibration by the plexor tympany is recognized The percussion note has a drumlike hollow quality distinct from normal resonance Tympany is normally heard over the *distended abdomen* It may also be detected over the larynx or trachea It is abnormal if it is elicited anywhere except over the larynx trachea or left lower ribs anteriorly

3 **Dullness and Flatness**—Dullness and flatness result from replacement

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CONTINUED

cracked pot amphoric and metallic notes (p 3540) The cracked pot sound is imitated by striking the loosely clasped hollow palms of the hands together against the knee

Auscultation.—Auscultation can be performed *directly* the ear of the practitioner resting on the chest of his patient or *indirectly* by means of the stethoscope

Direct Auscultation.—Direct auscultation is used altogether too infrequently It is an awkward procedure particularly in the case of obese female patients with heavy breasts It cannot be used in the supraclavicular regions Under other circumstances direct auscultation conveys a purity of sound that cannot be approached with the stethoscope Certain adventitious sounds (*subcrepitant rales* and the high pitched *diastolic murmur* of aortic insufficiency) are best heard by direct auscultation

Indirect Auscultation with the Stethoscope.—Most practitioners employ indirect auscultation with the binaural stethoscope The particular model employed is a matter of individual choice *Ear pieces* should be carefully selected A loose ear piece produces extraneous noises A tight ear piece may cause pain and result in irritation or furunculosis of the external auditory canal

Chest pieces are of the bell or diaphragm types The former is best adapted for examining the apex of the lung It conveys well the sound of low pitched murmurs The diaphragm type cannot be made to fit in the supraclavicular regions It transmits a greater intensity of sound and is best adapted for detecting higher pitched blowing murmurs Instruments with interchangeable chest pieces are highly recommended

The *rubber tubing* of the stethoscope must not be excessively long A length of 8 to 12 inches is sufficient

The Technic of Auscultation of the Lungs.—The position of the patient during auscultation of the lungs is the same as during percussion For the investigation of the *anterior* and *axillary* regions the patient should be recumbent the hands clasped over the head For the examination of the *supraclavicular* and *posterior* regions the patient sits upright Those who are too ill must be examined in the right and left lateral recumbent positions The findings in the dependent chest are the more reliable the signs in the upper chest often being misleading

Normal quiet respiration through the nose does not adequately bring out the breath sounds The patient is therefore instructed to breathe through the mouth and to emphasize expiration by a short huff as though a candle were being extinguished

During auscultation the physician compares corresponding points on the two sides of the chest always bearing in mind the modifications due to the presence of the heart on the left and the dome of the liver on the right It would be ideal if auscultation could be performed in a sound proof room but it is better training for the practitioner to learn auscultation in the midst of all manner of extraneous noises

The Results of Pulmonary Auscultation 1 Extraneous Sounds.—Quite aside from the noises heard from without extraneous sounds may be transmitted through the stethoscope These include (1) the friction of the examiner's finger against the stethoscope (2) the rubbing of the stetho-

of the alveolar air by an exudate or the interposition of solid or fluid material between lung and thoracic cage

The Results of Percussion—The Lungs—The anterior and axillary portions of the chest are, as has been said percussed with the patient recumbent hands clasped over the head. Resonance prevails throughout except for the areas of cardiac dullness and flatness liver dullness and stomach tympany

Percussion of the posterior region is begun at the superior margin of each lung. Since the pulmonary tissue extends 2 or 3 cm above the clavicle a band of resonance corresponding to the shoulder strap is delineated. This band measures 4 to 6 cm in breadth. During deep inspiration it widens appreciably if the apex is free. The apex of the right lung is less resonant than the left. The percussion of the posterior chest proceeds from above downward the corresponding point on the opposite chest being used as a norm or control. The regions beneath the scapulae may be explored by having the patient place each hand on the opposite shoulder.

At the bases of the lungs the respiratory excursion is measured by percussion during inspiration and expiration. The normal descent approximates 2 or 3 cm. The right base because of the proximity of the liver is slightly duller than the left.

The Heart Borders—With the apex impulse as a guide the cardiac borders are defined by percussion. Extremely light percussion is used for the delineation of the right border heavier percussion is necessary for the left border. The earliest change from resonance to dullness outlines the cardiac silhouette. The pleximeter finger should parallel the expected border.

The right border of relative dullness parallels the right border of the sternum. The left border begins approximately 2 cm from the midsternal line in the third interspace. It slants downward and outward to reach a point 8 to 10 cm from the midsternal line in the fourth fifth or sixth interspace. The percussion of the left border may be checked by palpation of the point of maximum impulse.

Dullness is normally heard in the chest over the left border of the heart. **Flatness** is normally heard at the left border of the sternum in the fifth or sixth interspaces where the pericardium lies just beneath the chest wall.

Absolute Cardiac Flatness—Complete flatness is present over that portion of the pericardium not covered by lung tissue. This zone is triangular and occupies a position just to the left of the sternum at the level of the fourth rib.

Liver Dullness—The upper border of liver dullness is detected below the fourth rib anteriorly and extends for approximately two interspaces where liver flatness is encountered.

Delineation of Spleen and Stomach—In the left axillary region an area of dullness corresponding to the spleen is sometimes described by experts. Here also may be outlined a semilunar area of tympany (Traube's space) corresponding to the stomach. The results of percussion in these areas are too variable for accurate interpretation.

Abnormal Percussion Sounds—Abnormal percussion sounds include

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|------------------------------|--|
| Increased precordial dulness | Cardiac dilatation and hypertrophy (p 968) Pleural and pericardial effusion (p 2219) Confirm physical signs with x ray of chest and Ecg Consider diagnostic thoracentesis or pericardiocentesis (p 852) |
| Decreased precordial dulness | Emphysema with diminution of breath sounds (p 2056) Bronchial asthma with broncho spasm relieved by epinephrine (p 2101) Mediastinal emphysema with crunching sensation (p 2082) Pneumothorax with diminution of breath sounds and ipsilateral displacement of mediastinum (p 2035) Confirm physical signs with x rays With urgent symptoms summon thoracic surgeon |
| Increased liver dulness | Hypertrophic cirrhosis of liver (p 1969) Liver abscess or neoplasm (p 1980) Ascites (p 1921) Subdiaphragmatic abscess Get liver function tests (p 1947) Remove ascitic fluid (p 1823) Transfer to operating room for aspiration of liver by surgeon |
| Decreased liver dulness | Tympanites (p 1878) Atrophic stages of acute liver atrophy (p 1968) Free air in peritoneal cavity due to perforation of hollow viscus (p 1790) Get abdominal x rays erect and recumbent Summon surgeon |

scope on the skin particularly over the hairy parts (3) muscle sounds produced when the patient is tense nervous or chilled and (4) noises due to the stretching of adhesive plaster or the pressure of clothes These auditory nuisances must be recognized and disregarded if they cannot be eliminated

2 Normal Auscultatory Sounds—There are three basic auscultatory sounds *Vesicular breathing* is heard over pulmonary alveoli *Bronchial* or *tubular breathing* occurs over the trachea and large bronchi *Broncho vesicular breathing* is a synthesis of tubular and vesicular breathing, and is heard where pulmonary alveoli lie in close proximity to larger bronchi

Auscultatory sounds have inspiratory and expiratory components and the intensity pitch and duration of each are noted

NORMAL VESICULAR BREATHING—The normal vesicular sound is heard most distinctly over the anterior chest in the axilla and posteriorly just below the scapula The normal vesicular sound must be heard to be appreciated The *inspiratory* component is more intense longer and pitched higher than the *expiratory* component The quality of the sound is said to be breezy and is best heard when the patient breathes with the mouth open The *expiratory* sound is much softer If it is heard with difficulty it may be accentuated by instructing the patient to give a short huff as if to extinguish a lighted candle

Vesicular breathing may be *increased* or exaggerated in children or adults with thin narrow chests It may be *distant* in those with heavy chests or *absent* under pathological circumstances *There are no conditions under which vesicular breathing is pathological*

DIFFERENTIAL DIAGNOSIS OF

***Abnormalities Noted by Percussion
of the Chest Wall***

Accurate percussion is of inestimable value in the determination of intrathoracic disease. In the home where radiography and other accessory methods of diagnosis are not easily available the practitioner must place greatest reliance upon alterations in the transmission of sound as determined by pleximetry and auscultation with the stethoscope.

PHYSICAL FINDING CAUSES AND DIAGNOSTIC FEATURES

Hyperresonance

Normal in the linear chest (p 3488) With suppressed high pitched breathing in early stages of lobar consolidation (p 2171) With diminution of breath sounds in emphysema (p 2066) With bronchospasm relieved by epinephrine in bronchial asthma (p 2101) With localized absence of breath sounds and contralateral mediastinal displacement in pneumothorax (p 2035) With amphoric breathing and cracked pot note in pulmonary excavation (p 2200) Confirm physical signs with x ray Get sputum for demonstration of pathogen (p 3720)

Tympany

With high pitched suppressed respiration in early stages of lobar consolidation (p 2171) With suppression of breath sounds and ipsilateral mediastinal displacement in pneumothorax (p 2035) With eventration of diaphragm and diaphragmatic hernia demonstrable by x ray and barium meal (p 1799) Confirm physical findings with x ray of chest Examine sputum for demonstration of pathogen (p 3720)

Dulness to flatness with increased breathing

Consolidation in lobar pneumonia (p 2171) Confirm physical signs with chest x ray Obtain sputum and blood for demonstration of pathogen (p 3720)

Dulness to flatness with decreased breathing

Pleural effusion with contralateral displacement of mediastinum (p 2219) Pulmonary atelectasis with ipsilateral mediastinal displacement (p 2052) Confirm physical signs with x ray Consider diagnostic thoracentesis (p 2222)

Coarse sound

Hydropneumothorax and large excavations containing air and fluid (p 2035) Confirm physical signs with chest x ray Check for succussion sounds

Increased retrosternal dulness

Dilatation or aneurysm of aorta (p 798) Aortitis (p 892) Dilated pulmonary conus (p 969) Persistence of thymus (p 1236) Neoplasm of thymus or mediastinum (p 1234) Mediastinal lymphadenopathy (p 2082) Intra-thoracic goiter (p 1220) Acute pericarditis with friction rub (p 1007) Cor bovinum (p 968)

Confirm physical signs with fluoroscopy and x ray of chest Get serology for syphilis (p 336) Obtain Ecg (p 803)

in the chest when a loop of bowel has herniated through the diaphragm as in cases of *eventration*

THE COIN SOUND—The coin sound is the equivalent of the metallic tinkle. It is produced by striking the edge of a plexor coin upon the surface of a pleximeter coin held firmly against the chest wall. The stethoscope of the auditor is placed on the chest wall opposite to the impinging coins. When the coin sound is positive the metallic tinkle will be heard in close proximity.

ADVENTITIOUS BREATH SOUNDS—Adventitious sounds heard during the examination of the normal chest may be due to friction from clothing or hair or from muscle tremors produced by tenseness or shivering. Normally adventitious sounds are not produced within the pulmonary alveoli. The pathological adventitious sounds of pulmonary origin are the *friction rub* and the various types of *rales*.

PLEURAL AND PERICARDIAL FRICTION SOUNDS—The pleural friction rub is easy to detect. Its sound may be simulated by rubbing the index finger on the tragus of the ear. Commonly the friction rub is localized, unilateral and heard equally in inspiration and expiration. It will not disappear with cough. It is usually associated with localized pain and tenderness. It may be palpable as well as audible. It is produced by a fibrinous exudate on a serous surface.

The rub of *pericarditis* (p. 1007) best described as a *shuffle* is differentiated from the pleural friction rub by its location and its independence of respiration. *Pleuropericardial* rubs may give rise to some difficulty of interpretation since they possess the features of both the pleural and the pericardial friction.

RALES—Rales are adventitious sounds produced in an airway, their characteristics varying with the type and site of the exudate present in the passage. (1) *Tracheal rales*. The most obvious rale is that heard with fluid in the upper trachea which is not unlike a gurgling sound. It is heard over the entire chest and constitutes the so-called death rattle. (2) *Bubbling rales* are coarse rhonchi produced by the passage of the air through a fluid exudate of serum, mucus, pus or blood in a large or medium sized bronchus. In generalized bronchitis these are heard all over the chest. In the presence of a localized lesion the sound will be limited to the lobe involved. (3) *Sonorous and sibilant rales*. Sonorous and sibilant rales as well as the *musical and whistling* varieties are heard when there is tenacious exudate, a foreign body in a large bronchus or stenosis or deformity of the bronchial wall. *Localized rales* of these types suggest a focal disturbance such as endobronchial tumor or foreign body. (4) *Crepitant and subcrepitant rales*. The most important of the rales are produced in the alveoli and terminal bronchioles. The *subcrepitant rale* occurs with exudate in a bronchiole. Its sound may be simulated by twisting the hair over the mastoid region. The *crepitant rale* is the only rale produced in the alveoli. It is pathognomonic of *parenchymal pulmonary disease*. It is a fine, high pitched rale heard chiefly at the end of inspiration. It is not affected by cough. Its sound may be simulated by separating the fingers moistened with saliva in close proximity to the ear.

Crepitant rales also known as *consonating rales* are the most important and the most difficult to recognize. They may be the earliest physical sign

TUBULAR OR BRONCHIAL BREATHING—Tubular tracheal or bronchial breathing is normally heard over the trachea larynx, upper portion of the sternum anteriorly and in the medial line posteriorly down to the level of the second, third or fourth spinous process. It is pathological when it is heard elsewhere.

Bronchial breathing is of increased intensity and higher pitch than vesicular breathing. The duration of expiration exceeds that of inspiration. The percussion note over the area of bronchial breathing is usually dull to flat. Bronchial breathing may be *distant* or *exaggerated*.

BRONCHOVESICULAR BREATHING—Bronchovesicular breathing is a mixture of the bronchial and vesicular elements. Either of the two qualities may predominate. Usually inspiration has more of the vesicular quality with a slight increase in intensity and an elevation of pitch. Expiration is slightly prolonged and partakes of the bronchial character. Normally bronchovesicular breathing is audible where alveoli overlie large bronchi. In health this condition prevails lateral to the median line in the upper sternal region anteriorly and in the upper thoracic region posteriorly. Bronchovesicular breathing is normal at the right apex anteriorly and posteriorly. Bronchovesicular breathing heard elsewhere than in the normal regions is an indication of a pathological state. The percussion note associated with bronchovesicular breathing is impaired resonance to dullness.

3 Abnormal Auscultatory Findings **ASTHMATIC BREATHING**—In asthmatic breathing the quality of the breath sounds may be unchanged. The duration of inspiration however is usually markedly reduced whereas expiration is unduly prolonged and high pitched. Almost invariably there are accompanying wheezing rales. The percussion note is hyperresonant or tympanic.

CAVERNOUS BREATHING—Cavernous breathing is always pathological. It occurs over a cavity or an area of pneumothorax. Its phonetic equivalent is the whispered word *who*. It is a deep hollow low pitched sound not unlike that produced by blowing into a test tube. The overlying percussion note is usually tympanic to cracked pot.

AMPHORIC BREATHING—Amphoric breathing resembles cavernous breathing. It is higher pitched and has more of a metallic quality. It is produced over a cavernous area whose tense walls impart a vibration to the sound produced in the cavity. The percussion note is tympanic to cracked pot.

COGWHEEL BREATH SOUNDS—In cogwheel respiration inspiration and expiration are interrupted by short irregular pauses. It is commonly heard over the upper lobes where it may be a normal finding. Cogwheel breathing may be an early physical manifestation of *incipient tuberculosis* when there are fine adhesions or sticky exudate interfering with the smooth ingress and egress of air.

METALLIC TINKLE—The metallic tinkle whose designation indicates its quality occasionally may be heard in circumstances where there are both free air and free fluid in the pleural cavity or within a pulmonary excavation.

SUCCUSSION SOUNDS—Succussion sounds are splashing noises that are heard when air and fluid are both present within an enclosed space such as a pulmonary, pleural or pericardial cavity. Succussion may

in the chest when a loop of bowel has herniated through the diaphragm as in cases of *eventration*

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DIFFERENTIAL DIAGNOSIS OF

***Abnormalities Noted by Auscultation
of the Chest Wall***

Direct and indirect auscultation of the chest discloses changes in inspiratory or expiratory sounds and pathological sound effects such as rales and friction rubs. Abnormalities of the cardiac sounds are described in the table on p. 778.

PHYSICAL SIGNS CAUSES AND DIAGNOSTIC FEATURES

| | |
|---|--|
| Increased expiratory phase | Emphysema with diminution of breath sounds (p. 2056) Bronchial asthma with bronchospasm, relieved by epinephrine (p. 2101) |
| Diminished respiratory sounds with hyperresonance | Emphysema with barrel chest (p. 2056) Pneumothorax with suppression of breath sounds and ipsilateral deviation of mediastinum (p. 2035) Eventration of diaphragm or diaphragmatic hernia with demonstrable intestinal content within thorax (p. 1779) Confirm physical signs with chest x ray & supplemented by barium meal |
| Diminished respiration with dullness to flatness | Pleural effusion, tumor or fibrosis Empyema thoracis (p. 2219) Acute or chronic pneumonitis (p. 2171) Pulmonary atelectasis (p. 2052) Pulmonary abscess or neoplasm (p. 2214) Hypostasis (p. 2086) Confirm physical signs with chest x ray Perform diagnostic thoracentesis when indicated (p. 2222) Obtain sputum for stain and culture |
| Bronchovesicular bronchial and tubular breathing | Pulmonary consolidation due to lobar pneumonia (p. 2171) acute pneumonitis (p. 2185) chronic pneumonitis (p. 2209) large bronhectasis (p. 2059) pulmonary abscess (p. 2214) atelectasis (p. 2052) foreign body impaction (p. 2075) or embolization (p. 2086) Supplement physical signs with x ray of chest (p. 3741) Stain and culture sputum (p. 3770) Consider bronchoscopy with lipiodol injection for contrast films (p. 2026) |
| Pectoriloquy | Pulmonary compression over fluid level (p. 2219) Supplement physical examination with chest x ray Consider diagnostic thoracentesis (p. 2222) |
| Amphoric and cavernous breathing | Pulmonary cavitation or abscess (p. 2052) Supplement physical examination with chest x ray sputum examination and bronchoscopy (p. 2026) |
| Egophony | Over area of compression above pleural effusion (p. 2219) Confirm finding with chest x ray Consider diagnostic thoracentesis (p. 2222) |

| | |
|--|---|
| SIGNS | Hydropneumothorax or large cavitation containing air and fluid (p 2035) Eventration of diaphragm or diaphragmatic hernia (p 1799) Supplement physical examination with chest x ray and sputum examination (p 3120) Give barium meal for demonstration of intestinal viscera within thorax (p 1799) Consider diagnostic thoracentesis |
| Cogwheel breathing at apex | Early tuberculous infiltrations (p 2199) Obtain temperature record. Get sputum and gastric contents for tubercle bacilli (p 3720) Get x ray of chest. |
| Sibilant or sonorous rales and rhonchi | Bronchial asthma with bronchospasm relieved by epinephrine (p 2101) Emphysema with distant breath sounds (p 2036) |
| Generalized bubbling rales | Pulmonary edema (p 2086) Acute bronchitis (p 2165) |
| Generalized crepitant rales | Acute and chronic bronchitis (p 2168) Confirm physical signs with chest x ray and sputum examination Obtain temperature record (p 3484) |
| Localized crepitant rales | Acute and chronic bronchitis (p 2165) influenza lobar pneumonia (p 2171) acute and chronic pneumonitis (p 2209) pulmonary abscess (p 2214) bronchiectasis (p 2059) atelectasis (p 2052) foreign body impaction (p 2075) embolization (p 2086) and tuberculous infiltrations (p 2199) Confirm physical signs with chest x ray sputum examination and bronchoscopy if indicated. Get temperature record (p 3484) |
| Subcrepitant rales | Lobar pneumonia acute pneumonitis and tuberculous infiltrations Confirm physical signs with sputum examinations and chest x ray Get temperature record (p 3484) |
| Friction rub | Fibrinous pleuritis or pericarditis (p 1007) Note relief from immobilization |

of parenchymal disease particularly in *tuberculosis* (p 2199) The commonest places for them to be heard under these circumstances are the infraclavicular or the supraclavicular regions on the right side

Method of Eliciting Rales—*Latent rales* particularly in early tuberculosis may be brought out when the patient breathes more deeply particularly stressing expiration The so-called *post tussive rale* is elicited by interpolating a cough at the end of expiration In tuberculosis when the exudate is slight the rale may be heard only with the first breath The area under suspicion is best examined immediately after the patient has arisen The suspected site should be examined before investigating the rest of the pulmonary field

4 Voice Sounds (Whispered and Spoken)—The registration of the sounds of the whispered and spoken voice complement the information that is obtained from auscultation of the breath sounds Commonly the *whispered voice* is tested by having the patient say one two three the

spoken voice by repeating ninety nine in the lowest range of the speaking voice

VOCAL RESONANCE BRONCHOPHONY AND PECTORILOQUY—The whispered and spoken voice give information that is similar to that obtained from the breath sounds. *Normal vocal resonance* corresponds to the vesicular sound. *Bronchophony* and *pectoriloquy* whispered or spoken correspond to bronchovesicular and bronchial breathing.

EGOPHONY—*Egophony* is an additional sound like the bleating of a goat. It is usually heard near the upper level of a pleural effusion. It is probably produced in the compressed lung that lies above the fluid.

FREMITUS—The normal vocal sounds may be palpated as well as heard. The resulting *fremitus* is best detected by the tips of the fingers. Vocal fremitus is most readily obtained in the male because of his deep voice. The patient speaks in a uniform low pitched voice. *Fremitus* is compared over symmetrical areas of the chests. It may be absent, increased or diminished.

5 Chest Aspiration for Diagnostic Purposes—The physical signs that suggest the presence of fluid in the free pleural cavity include flatness, decreased fremitus, diminished to absent breath sounds and displacement of the mediastinal contents to the opposite side. Under these circumstances it is often necessary to corroborate the physical findings by *chest aspiration*. In this way both the presence of fluid and its physical characteristics are noted.

The technic of diagnostic chest aspiration is similar to that of *thoracentesis* (p 2222) except that the needle is introduced while attached to the barrel of a 20 cc syringe. Only sufficient fluid is withdrawn for diagnostic purposes. In the presence of large amounts of fluid the *Potain set* (p 2030) is held in readiness. Its connecting piece may be attached to the needle as soon as the syringe is disconnected. This saves the patient separate procedures for diagnostic aspiration and therapeutic evacuation.

The fluid withdrawn from the chest is carefully examined. The first portion is collected aseptically in a sterile test tube and sent to the bacteriologist for *culture*. A second sample is examined for the number of cells and the specific gravity. Material is also spread on slides to be stained with (1) *methylene blue* for the general character of the cells, (2) *Wright's stain* for the differential leukocyte count, (3) *the Gram method* in the hope that the identity of the invading organism may be suggested for purposes of surgical indication and/or chemotherapy.

With large pleural effusions the entire yield of the aspiration is sent to the pathologist for *histologic study*. Bloody fluids particularly should be dealt with in this way as it is sometimes possible to recognize tumor cells. See *Differential Diagnosis of Pleural Effusion* (p 2032) *Pneumothorax* (p 2035).

6 Diagnostic Lung Puncture—The discomfort and danger of lung puncture disqualify this diagnostic measure for use by the practitioner. Lung puncture is justified when there is the suspicion of a *neoplasm* (p 2081) or an *abscess* (p 2214). Material obtained is examined microscopically and histologically as in a biopsy (p 3935).

Diagnostic lung puncture is conducted in the same manner and with the same apparatus as diagnostic chest aspiration. The needle is thrust

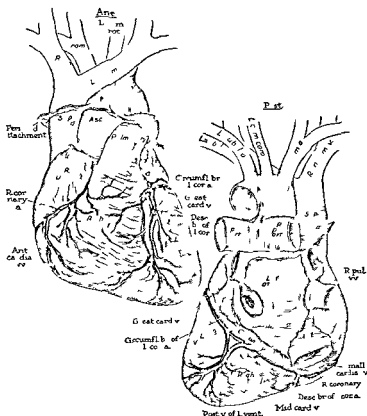
deeply into the lung tissue in the attempt to obtain alveolar contents or a specimen of lung tissue

THE BLOOD VASCULAR SYSTEM

ANATOMIC REVIEW

The blood vascular system consists of the heart and blood vessels. The heart situated in the thorax is contained in the pericardium, a conical serofibrous sac.

The **Pericardium**—The pericardium placed in the middle mediastinum lies on and is attached to the diaphragm. It consists of an external fibrous pericardium and an internal serous pericardium. Its base is applied to the diaphragm where it is pierced by the inferior



from the fifth to the eighth inclusive. The *apex* is directed downward forward and to the left and corresponds to the space between the cartilages of the fifth and sixth ribs approximately 8 cm from the midsternal line.

The *anterior portion* of the heart is composed almost entirely of right ventricle. A small portion of the left ventricle makes up the apex while the right auricle forms the right border. The under portion of the heart which rests upon the diaphragm is formed by the ventricles chiefly the left. The base of the heart is formed by the auricles. To the right side the superior and inferior cavæ enter from above and below. The left ventricle forms the left border of the base being separated from the left auricle by the coronary sinus. The right margin of the heart is long and is formed by the right auricle. The left margin is formed exclusively by the left ventricle except to a slight extent above where the tip of the left auricle may be seen.

The Cavities of the Heart—The cavity of the heart is subdivided by a muscular septum into a *right pulmonary heart* and a *left or systemic heart*. A transverse constriction subdivides each of these main cavities into a posterior cavity or *auricle* and an anterior cavity or *ventricle*. The heart consequently contains four chambers.

The *cavities* of the heart are lined by endocardium which is reduplicated at the ostia to form valves. The *mitral* separating left auricle and ventricle and the *tricuspid* the right auricle and ventricle. As the names imply the mitral valve is made up of two and the tricuspid valve of three leaflets.

1 The *right auricle* is the larger and consists of a principal cavity the *sinus venosus*, situated posteriorly and a smaller *auricular appendix* situated anteriorly. The *sinus venosus* is formed by the two *venæ cavæ* returning the venous blood from the systemic channels. Into the right auricle also flows blood from the *coronary sinuses* and the *foramina Thebesii*—the latter bringing the blood directly from the muscle substance of the heart. The right auricle discharges its contents through the tricuspid orifice into the right ventricle.

2 The *right ventricle* forms most of the anterior portion of the heart. It is situated somewhat transversely. Its under surface rests upon the diaphragm. The posterior wall is formed by the interventricular septum separating it from the left ventricle. The upper and inner angle of the right ventricle is prolonged into a conical pouch the *conus arteriosus* or *infundibulum* from which the pulmonary artery arises separated from the cavity of the right ventricle by the three cusps of the pulmonary valve. The walls of the right ventricle are thinner than those of the left—the proportion being as one is to three. Because of the higher interventricular pressure in the left ventricle the septum bulges into the right ventricle under normal conditions.

The right auriculoventricular opening or *tricuspid orifice* is situated nearly in the vertical plane near the right border of the heart just above the diaphragm. It is oval and about 1½ inches in diameter. Normally it will admit the ends of four fingers. It is guarded by the *tricuspid valve* which consists of three leaflets—a left right and posterior segment. Each segment is connected by its base to the oval fibrous ring surrounding the orifice. The cusps are connected to each other by their sides. Their serrated free margins afford attachment to the chordæ tendineæ which are attached to the papillary muscles of the ventricular wall. The wall of the ventricle contains the *columnæ carneæ* projections due to the rounded muscle columns. These form ridges arches or papillary muscles. Occasionally a moderator band stretches across the cavity. The opening of the *pulmonary valve* is circular in form. It is situated above and to the left of the tricuspid orifice.

3 The *left auricle* is smaller than the right but the walls are thicker. It consists of a left auricular appendage which forms a small portion of the upper left cardiac border and a principal cavity which lies behind the pulmonary artery and aorta. In front and to the right it is separated from the right auricle by the interauricular septum. Behind it receives on either side the pulmonary veins. The principal cavity of the auricle presents the orifices of the four pulmonary veins that open into the upper part of the posterior surface.

The auricle communicates with its ventricle by the *mitral orifice* which is horizontally situated and placed below and to the left of the aortic opening. It is slightly smaller than the tricuspid orifice admitting only two fingers. It is guarded by the *mitral valve* which has an anterior or aortic and posterior or marginal leaflet. These leaflets are larger thicker and stronger than those of the right heart. The cusps are provided with chordæ tendineæ as on the right side. The inner surface of the left auricle also contains the *foramina Thebesii*.

4 The *left ventricle* forms a major part of the posterior and inferior portion of the heart. It constitutes the apex by its projection anteriorly beyond the right ventricle. Its

walls are thicker than those of the right side the proportion being as 3 is to 1. The blood from the left auricle passes from above downward through the mitral orifice into the ventricle. The walls of the ventricle contain columnae carneae as on the right side.

Blood from the left ventricle is propelled through the aortic opening which is circular and situated in front and to the right of the mitral orifice. The aortic orifice is guarded by the aortic cusps which consist of three semilunar segments.

The interventricular septum separates the two ventricles. The greater portion of the septum is thick and fleshy. The upper portion is thin and fibrous and is termed the "undeflected portion." See *Diagnostic Roentgenology of Cardiac Contour* (p. 292) Figs. 119 to 128 (pp. 292-298).

The Size and Weight of the Heart.—The normal heart in the adult measures 12 cm. in length, 9 cm. in breadth and 6 cm. in thickness. The weight in the male varies from 300 to 350 gm. and in the female from 240 to 300 gm. Each of the cavities of the heart is capable of holding about 100 cc. See *Differential Diagnosis of Cardiac Hypertrophy* (p. 865).

The Structure of the Heart.—The heart is a hollow muscular organ the walls of which have three coats: the serous *endocardium*, the *myocardium* and the visceral layer of the pericardium.

THE MYOCARDIUM.—The myocardium is made up of superficial and deep muscle layers of the auricles and ventricles and a specialized *auriculoventricular bundle* which bears the name of the anatomist His. This bundle arises near the opening of the coronary sinus. In the right auricle the fibers converge to form the *node of Archoff-Tawara* and they continue as a compact bundle passing forward in the lower portion of the undeflected space of the interventricular septum to the upper limit of the muscle portion of the septum. There the bundle divides into a right and left segment thence spreading over the entire internal surface of the ventricular muscle and forming histological connections (Purkinje fibers) with the true cardiac muscle fibers (Fig. 119).

Surface Form.—The *pulmonary orifice* is situated at the junction of the third left costal cartilage with the sternum. The *aortic orifice* is slightly below and to the right of the pulmonary opening occupying a position almost in the midsternal line. The *mitral orifice* is situated just below the aortic valve at the junction of the fourth costal cartilage and the sternum. The *tricuspid valve* horizontally placed extends in the midsternal line from the level of the junction of the fourth costal cartilage and the sternum to the level of the junction of the fifth costal cartilage and the sternum.

The *apex impulse* is commonly placed approximately 8 cm. from the midsternal line in the left fifth or sixth interspace. The area of *relative cardiac dulness* extends about 1 cm. to the left of the point of maximum impulse. The area of *complete cardiac dulness* extend to within 2 to 3 cm. of the point of maximum impulse. The *left border* of the heart forms a convex line from the area of maximum dulness in the fifth or sixth interspace to the junction of the second costal cartilage and the sternum. The *right border* of the heart forms a convex line that extends 1 or 2 cm. to the right of the midsternal line from the junction of the second costal cartilage and the sternum to the junction of the fifth costal cartilage and the sternum.

Blood Supply to the Heart.—The blood supply to the heart is mainly through the *coronary system*. The coronary arteries arise near the commencement of the aorta immediately above the attached margin of the semilunar valves. The *right coronary artery* passes forward between the pulmonary artery and the right auricular appendage and divides into a transverse branch that passes in the groove between the left auricle and ventricle anastomosing with the left coronary through small channels and a descending branch which courses along the interventricular furrow supplying branches to both ventricles and the septum (Fig. 1027).

The *left and left coronary artery* passes forward between the pulmonary artery and the left auricular appendage and divides into a transverse branch which passes outward in the left auriculoventricular groove and a descending branch which passes along the interventricular groove to anastomose with the right coronary.

The coronary veins follow the course of the coronary arteries but join to form a coronary sinus about an inch in length that terminates in the right auricle. In addition three *left anterior cardiac veins* open directly to the right auricle as do the *veins of Thebesius*. The latter empty blood directly into both auricles and ventricles.

Cardiac Nerves.—The nerves of the heart are derived from the *superficial and deep cardiac plexuses*. The former lies under the arch of the aorta the latter in front of the

bifurcation of the trachea. The plexuses obtain fibers from the vagus, the spinal accessory and the sympathetic chains. Adrenergic fibers on stimulation produce tachycardia while cholinergic effects lead to bradycardia and so-called "vagus arrest."

THE TECHNIC OF CARDIAC AUSCULTATION

For *direct auscultation* the patient is first examined while recumbent then sitting up and bent forward so that the chest rests on the listening ear of the examiner. Direct auscultation may reveal an *aortic diastolic murmur* audible through the stethoscope.

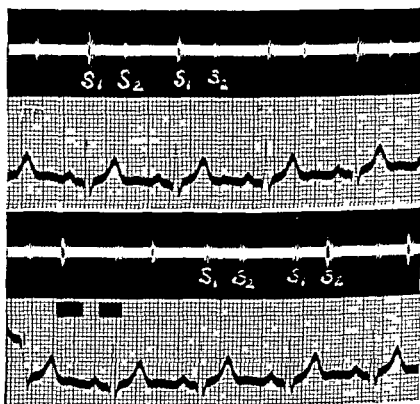


Fig 1098—Normal heart sounds. Simultaneous phonocardiograms and electrocardiograms of a normal male 29 years of age. The upper sound curves were taken from the apex and the lower from the pulmonary area. Note that the first sound (S_1) is louder at the apex and fainter at the base than the second sound (S_2). The relative intensity of the two sounds varies both in normal and abnormal hearts.

For *indirect auscultation* the patient is examined in several positions: sitting erect, lying recumbent, and in the right and left lateral decubitus. The last position is stressed in suspected *mitral stenosis* (p 973) since the murmur otherwise may be inaudible.

THE RESULTS OF CARDIAC AUSCULTATION

The Normal Cardiac Sounds at the Apex—At the apex the examiner normally hears a first and second sound. The *first sound* is produced mainly by the vibrations of the heart walls and valves. It corresponds with systole.

• Levine Clinical Heart Disease

and is described as muscular in quality. The *second sound* results from the sudden increased tension and subsequent vibrations during closure of the aortic and pulmonary leaflets. The second sound is heard with the onset of diastole. It is sharper, more concise and may be described as valvular in quality.

In young people with narrow chests there is in addition to the two normal cardiac sounds a *third apical sound* which can occasionally be heard with the patient in the left lateral decubitus. The sound is much like an echo of the second sound. It has no clinical significance.

The Normal Cardiac Sounds at the Base—At the base of the heart auscultation is carried out over the aortic and pulmonary areas. In either situation two heart sounds are audible, each possessing the valvular quality of the second apical sound. Until adolescence the *pulmonary second sound* is the louder. After adolescence and throughout the rest of the normal life the *aortic sound* is the more intense. See *Differential Diagnosis of Alterations in Normal Cardiac Sounds* (p. 778).

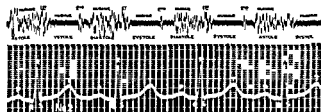


Fig. 10-9—Mitral stenosis. Stethogram at apex, electrocardiogram Lead II. Boy 8 years of age. Chorea at 4 years and again at 6 years. Probably low grade rheumatic fever since 6 years. Pneumatic heart disease known since 5 years. Hospital examination showed moderate systolic and moderate crescendo diastolic murmur. Latter accompanied by a thrill. The stethogram shows both murmurs clearly.

Abnormal Cardiac Sounds—Abnormal cardiac sounds consist of *friction rubs*, *reduplications* and *murmurs*.

The Friction Sounds—The *pericardial shuffle* or *pericardial rub* may be felt and heard. It implies a fibrinous exudate on the serous lining membranes.

Reduplications—Reduplications of the heart sounds are not necessarily pathological. At the apex the reduplication of the first sound is probably due to delayed contraction of the papillary muscles. Reduplication of the *second apical sound* probably results from asynchronous closure of the valves, suggesting *mitral valvulitis with stenosis*. The reduplication may precede the valvular murmur.

In instances where the ventricular rate is very slow the normal ventricular sound may be preceded by a faint *auricular sound*. See *Heart Block* (p. 879).

Reduplications of the basal sounds have the same implication as increases in intensity. They are probably due to asynchronous closure of the aortic and pulmonic valves.

Murmurs—Cardiac murmurs always require investigation. They differ from normal heart sounds in several respects. The normal sound is usually

bifurcation of the trachea. The plexuses obtain fibers from the vagus, the spinal accessory and the sympathetic chains. Adrenergic fibers on stimulation produce tachycardia while cholinergic effects lead to bradycardia and so-called "vagus arrest."

THE TECHNIC OF CARDIAC AUSCULTATION

For *direct auscultation* the patient is first examined while recumbent then sitting up and bent forward so that the chest rests on the listening ear of the examiner. Direct auscultation may reveal an *aortic diastolic murmur* inaudible through the stethoscope.

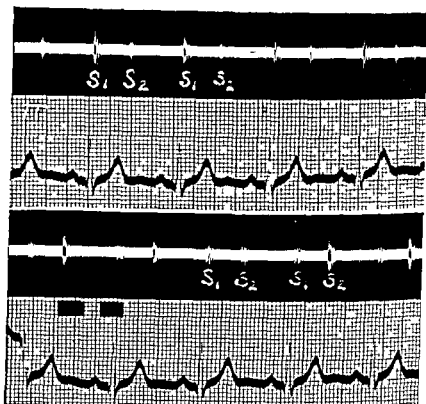


Fig. 1078.—Normal heart sounds. Simultaneous phonocardiograms and electrocardiograms of a normal male 29 years of age. The upper sound curves were taken from the apex and the lower from the pulmonary area. Note that the first sound (S_1) is louder at the apex and fainter at the base than the second sound (S_2). The relative intensity of the two sounds varies both in normal and abnormal hearts.

For *indirect auscultation* the patient is examined in several positions sitting erect, lying recumbent, and in the right and left lateral decubitus. The last position is stressed in suspected *mitral stenosis* (p. 973) since the murmur otherwise may be inaudible.

THE RESULTS OF CARDIAC AUSCULTATION

The Normal Cardiac Sounds at the Apex.—At the apex the examiner normally hears a first and second sound. The *first sound* is produced mainly by the vibrations of the heart walls and valves. It corresponds with systole.

* Levine: Clinical Heart Disease.

the third space to the left of the sternum *pulmonic murmurs* in the second left intercostal space and *tricuspid murmurs* over the lower part of the sternum

The *quality* and *intensity* of the murmurs vary from the *soft diastolic blow* heard along the left border of the sternum in aortic insufficiency to the *rough crescendo* rumble of a typical mitral stenosis. The latter is usually palpable as well as audible and is recognized as the presystolic apical thrill

The *timing* of the murmur places it in relation to systole or diastole. The *systolic murmur* at the apex denotes valvular insufficiency. At the base it indicates a stenosis. The *diastolic* or *presystolic murmur* at the apex is the stenotic murmur whereas at the base it occurs with insufficiency

Continuous murmurs are heard throughout all cycles of the heart beat. If they are shuffling they indicate a fibrinous pericarditis. Otherwise they point to some type of lesion usually congenital in which there is an arteriovenous shunt or an abnormal communication between the cavities of the heart such as a patent ductus arteriosus (p 957)

When the heart rate is rapid it is difficult to time the murmur. In this circumstance its mechanism of production may be suggested by the *attendant phenomena*. Thus a high pulse pressure suggests an aortic insufficiency. One that is exceedingly low occurs with aortic stenosis. See Table 63 Classification of Murmurs (p 973)

Alterations in Cardiac Rhythm—The normal rhythm of the heart is regular. Occasionally there may be a physiological variation consisting of a periodic increase in rate during inspiration and a decrease with expiration. This condition of *sinus arrhythmia* (p 877) is of vagal origin and commonly occurs in infants and young children. The presence of any other arrhythmia requires investigation particularly with the aid of electrocardiography. See *Cardiac Arrhythmias* (p 873)

loudest at the onset and gradually dies out (*diminuendo*) The murmur sustains the same degree of intensity for some time and even becomes louder (*crescendo*) It usually lasts longer than a normal sound Loud murmurs are commonly accompanied by palpable thrills Intracardiac murmurs must be differentiated from extracardiac sounds (*pericardial* and *pleuropericardial* rubs)

The cardiac murmur is produced in one or more of three ways (1) From *vibrations* set up as the blood rushes through a narrowed thickened valvular orifice (as in mitral and aortic stenosis) (2) from the *back leak* of blood through incompetent valves (mitral and aortic insufficiency), and (3) as the result of *swirls* or *currents* set up in the cavities of the heart with or without valvular defect

In the first two instances the murmur indicates a structural defect in the heart valves or in the cardiac septa and is termed a *true organic murmur* In the last instance the murmur may have no pathological sig

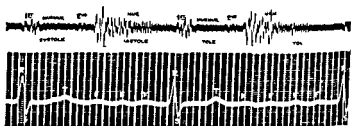


Fig 1030—Decrescending diastolic murmur Stethogram at left sternal border electrocardiogram Lead II Boy 16 years of age Rheumatic fever dates from 11 years Two attacks since then last beginning at 15 years Rheumatic heart disease known since 12 years Auricular fibrillation of unknown duration (several months at least) Hospital diagnosis showed systolic murmur beginning immediately after first sound long flowing diastolic murmur beginning promptly after second sound A marked thrill accompanies the diastolic murmur Note especially the easily recognizable decrescendic murmur shown by the gradually decreasing height of the deflections and the recording of the low pitch thrill by the wide spacing of the adjacent peaks of the diastolic murmur *

nificance under which circumstance it is called *functional hemic or accidental*

The examiner determines the nature of each murmur and evaluates its significance from the standpoint of cardiac function A *hemic murmur* is reported to the patient with the admonition that it is to be ignored An *organic murmur* must be identified for the correct management of the individual patient The *mitral insufficiency murmur* is of relatively little importance in the dynamics of the circulation However *aortic or tricuspid insufficiency* (p 973) and the *stenoses* (p 973) usually are or will be potent factors in circulatory difficulties

CHARACTERISTICS OF CARDIAC MURMURS—The important characteristics of cardiac murmurs are their *intensity quality timing* and the presence of *attendant phenomena* See Table 62 p 970

A murmur is usually best heard over the area where the corresponding heart sounds are most intense : *e* *mitral murmurs* at the apex *aortic murmurs* in the second intercostal space to the right of the sternum or in

abdominal wall become prominent and are to be distinguished from engorged vessels of collateral circulation. In portal obstruction the collateral circulation (between superior and inferior epigastric vessels) may line the abdominal wall with prominent venous channels.

The Muscle Layers.—Beneath the panniculus of the abdominal wall lie the firm muscle layers. Obese flabby patients, particularly multipara, may have a marked diastasis of the recti. Herniation occurs through the linea alba.

In the epigastric region small hernias may contain omentum or gut, under which circumstances the symptoms of organic intra-abdominal disease are simulated. Scars of abdominal incisions often become weakened, particularly if it has been necessary to employ drainage for any length of time. An incisional hernia may result.

To test for the various types of diastasis and herniation the patient is requested to tense the abdominal wall by raising the head and neck while lying otherwise recumbent. The suspected area is palpated with the muscles taut. Any point of tenderness or irregu-

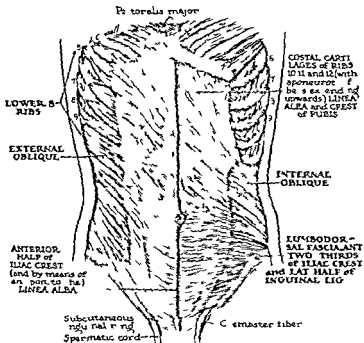


Fig. 1031.—External oblique and internal oblique muscles.

larity or actual sac is then demonstrated. The examination is repeated with the patient standing, the intra-abdominal pressure being raised by a cough.

The Inguinal Canal.—The examination of the abdominal wall anteriorly is completed by palpating the inguinal regions. The inguinal canals extend from the subcutaneous to the internal inguinal rings. The subcutaneous ring is situated 1 cm. above and lateral to the pubic tubercle. The internal ring lies 1 to 2 cm. above the middle of the inguinal ligament.

The rings and the canal are palpated for weakness or irregularity with the abdomen relaxed and taut. The examination is performed with the patient recumbent and then standing. The femoral rings are also tested at this time.

Herniation is best observed with the patient standing. A cough is employed to demonstrate the existence of a hernia.

Posterior Wall.—The posterior abdominal wall is formed by the vertebral column and the lumbar muscle groups. Its description accompanies the section on the Trunk (p. 309). Asymmetry in the lumbar region is noted.

* Mullard and King: Human Anatomy and Physiology.

CHAPTER 160

PHYSICAL EXAMINATION ABDOMEN

EXAMINATION of the abdomen resolves itself into the investigation of (1) the *abdominal wall* and (2) the *abdominal cavity* and its contents

PALPATION

The recognition of intra abdominal disease depends to a great degree upon palpation an art demanding sensitivity in the registration of the findings and in their interpretation The practitioner is required to visualize the anatomical details and relationships in order to transmute the information imparted by his finger tips into clinical gold To obtain the maximum data the examiner seats himself comfortably at the side of his patient, who must also be completely relaxed Palpation is then conducted in an orderly and leisurely manner with the attention of the patient distracted by questioning and conversation

The hands of the examiner must be warm and sudden or painful stimuli avoided Gentle steady pressure increased stealthily for the palpation of the deeper structures yields the best results The hand is first placed in the left lower quadrant the least likely site of difficulty and this region is tested first to gain the confidence of the patient and give a general idea of the normal tonus and feel of the wall The hands are then shifted to the left upper quadrant and the right side is felt last

In the patient who is incompletely relaxed the value of palpation is often greatly enhanced by the application of heat to the abdomen or by conducting the examination with the patient in a warm tub bath

THE ABDOMINAL WALL

ANATOMIC REVIEW

The Skin—The skin of the abdominal wall is thin In the female hair covers only the pubic region The male has a triangular growth of pubic hair whose apex is at the umbilicus

The fold of the groin on each side is the site of the *inguinal ligaments* Extending from the umbilicus to the symphysis pubis is the *linea alba* which becomes the pigmented *linea nigra* of pregnancy Lateral to this may be seen *striae* which are pigmented during pregnancy and which become atrophic scars when the abdominal wall has been restored to normal These striae result from rupture of elastic fibers when the abdominal wall is stretched by an increase in intra abdominal pressure They are not specific to pregnancy and may occur in the male or female

The Bony Landmarks—The bony landmarks of the abdomen above are the *xiphoid process* and the free borders of the lower ribs below the *symphysis pubis* in the middle and the *anterior superior iliac spines* and the *iliac crests* laterally

The Umbilicus—The umbilicus marks the central point of the anterior abdominal wall Normally it is firm and dry as well as free from odor

The Panniculus—The panniculus is composed of the subcutaneous fat of the abdominal wall In extreme instances the panniculus may hang down like an apron

The Vessels—Normally prominent vessels are not visible or palpable in the abdominal wall When the skin is delicate and thin as in infancy the blue streaks of small venules can be made out Occasionally also in marked loss of weight for any reason veins of the

Asymmetry in abdominal respiration may be due to paralysis of the abdominal muscles. It is particularly important to observe this in the investigation of a febrile child during the course of an epidemic of *polio myelitis* (p 457).

Generalized weakness of the abdominal wall may be demonstrated by having the patient stand. In this position the weakened area protrudes.

THE SUPERFICIAL ABDOMINAL REFLEXES

The superficial abdominal reflexes are elicited in the four quadrants by stroking the skin with the head of a pin or the nail of the examining hand. The *normal response* is a contraction of the underlying abdominal muscles and a deviation of the umbilicus to the side of the stimulus. The response is generally more active in the upper quadrants. The reflexes are not present in young infants. They are difficult to elicit or absent in the patient with an obese or lax abdominal wall. Of greater significance is unilateral loss of reflex.

The *cremasteric reflex* consists in retraction of the scrotum and elevation of the testis when the inner aspect of the thigh is stimulated by light stroking. Unilateral loss of this reflex is of diagnostic importance.

ABDOMINAL PAIN AND TENDERNESS

Under normal circumstances the abdominal examination should not give rise to pain or tenderness, although the abdominal aorta and spastic viscera may be sensitive in the thin patient.

Significant abdominal pain and tenderness may be superficial or deep. The former constitutes *hyperalgesia of the skin*. Hyperalgesia, as differentiated from deep abdominal pain, is produced when the skin is pinched or stroked with a pinhead. It may be absent or insignificantly increased with deep pressure by the examining fingers. *Deep abdominal pain and tenderness* are manifestations of the gravest importance. They require careful investigation and immediate attention. See *Differential Diagnosis of Generalized Abdominal Pain* (pp 1748-2730), *Epigastric Pain* (p 1788), *Pain in Hypogastrium* (p 2302), *Left Lower Quadrant* (p 1866), *Left Upper Quadrant* (p 1942), *Right Lower Quadrant* (p 1880), *Right Upper Quadrant* (p 1959), *Umbilical Region* (p 1887), *Lumbar Pain* (p 2274), *Special Causes of Abdominal Pain in Pregnancy* (p 2662).

REBOUND AND PERCUSSION TENDERNESS

Direct pain and tenderness are elicited by pressure from the examining fingers. The production of tenderness when the pressure of the examining finger is released is termed *rebound tenderness* (p 1923). The sign often indicates peritoneal inflammation or irritation. It may also occur over distended or irritated bowel with intact peritoneum. Hence it is a valuable but not a pathognomonic sign of peritonitis.

Percussion tenderness suggests intra abdominal inflammatory disease with peritoneal involvement.

Tenderness in the flank particularly if unilateral is highly suggestive of *suppuration* or *calculus formation in the kidneys*. In examining for tenderness the physician strikes the region of the *costovertebral angle* with the ulnar edge of the palm. Acute tenderness that may be missed on firm palpation can be elicited by this maneuver. See *Pain Lumber* (p 2274).

Peritoneum—Beneath the abdominal wall lie fascia, preperitoneal fat and peritoneum. The peritoneum is a large mesothelial membrane which lines the abdominal wall as the *parietal peritoneum*. It is reflected upon the various organs to form the *visceral peritoneum*. Sensation is present only in the parietal layer. Between the two layers of peritoneum is the peritoneal cavity.

Above and connected to the stomach is a peritoneal diverticulum known as the *omental bursa* (*lesser peritoneal sac*). This communicates with the main cavity by the *foramen of Winslow*. Reflected from the lower surface of the stomach is the *greater omentum*, a fold of peritoneum which extends apron like over the abdominal viscera.

The peritoneal surfaces are normally smooth, glistening and closely apposed so that the cavity is merely potential.

THE TONUS OF THE ABDOMINAL WALL

Under normal circumstances the patient relaxes sufficiently so that the abdominal wall is soft and pliable. This facilitates palpation of the contents of the abdominal cavity.

Voluntary Rigidity—In patients who are ticklish abdominal palpation is often made difficult by voluntary rigidity. Under these circumstances the wall must not be stroked. The fingers are carefully placed on the site of palpation, then lifted and replaced on the next site. To distract attention the patient is requested to open the mouth and breathe deeply. If this is insufficient to overcome the voluntary rigidity the patient is placed in a warm bath and palpation is resumed under water.

Involuntary Rigidity—Involuntary rigidity of the abdomen is one of the most important physical signs. To obtain it accurately the patient must be thoroughly relaxed and assured that the purpose of the examination is not to produce pain.

Involuntary rigidity almost without exception points to the presence of *peritoneal irritation*. Once the examiner is assured of the presence of involuntary muscle rigidity it is a grave responsibility to determine the cause of the peritoneal irritation and then proceed with the therapeutic management which will in many instances require *diagnostic abdominal puncture* or *laparotomy*.

See *Differential Diagnosis of Involuntary Rigidity of the Abdominal Wall* (p 1746).

THE MOVEMENT OF THE ABDOMINAL WALL

The abdominal wall should be symmetrical. *Asymmetry* requires investigation. Fulness in the epigastrium usually denotes a dilated stomach and in the hypogastrium a distended bladder or an enlarged or gravid uterus. The swelling or asymmetry may be due to some abnormal growth.

Normally the abdomen distends slightly in inspiration and retracts slightly in expiration. The amount of abdominal respiration varies in different individuals; it is greatest in those who have fixation of the thoracic cage (emphysema) or when there is air hunger (circulatory insufficiency or pneumonia).

Asymmetry in abdominal respiration may be due to paralysis of the abdominal muscles. It is particularly important to observe this in the investigation of a febrile child during the course of an epidemic of *polio myelitis* (p 457)

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Percussion tenderness suggests intra abdominal inflammatory disease with peritoneal involvement.

DIFFERENTIAL DIAGNOSIS OF

Disturbances of the Abdominal Wall

Because of the greater interest of the clinician in disturbances of the abdominal cavity and its contents examinations of the more obvious structures of the abdominal wall are often neglected. Nevertheless these superficial derangements often have important diagnostic connotations.

PHYSICAL SIGNS CAUSES AND DIAGNOSTIC FEATURES

| | |
|---|---|
| Dermatoses | See p 3368 |
| Female hair escutcheon in the male | Dislocation of androgen-estrogen ratio (p 2527) Hypogonadism hypopituitarism, hypopituitarism and hypothyroidism See Feminization of the Male (p 2481) |
| Male hair escutcheon in the female | Dislocation of androgen-estrogen ratio (p 2527) Hypogonadism Pituitary basophilism Adrenal cortical neoplasm Arrhenoblastoma See Masculinization of the Female (p 2481) |
| Pigmentation | Linea nigra in pregnancy (p 2642) Adrenal cortical deficiency Hyperthyroidism hemochromatosis Pellagra See Disturbances of Pigmentation (p 3242) |
| Striae | Obesity Pregnancy with uterine enlargement Pituitary basophilism with masculinization and hypertension (p 1159) |
| Retraction of Wall | |
| Metabolic | Dehydration due to persistent vomiting (p 1772) and diarrhea (p 1840) particularly in infancy |
| Increased intracranial tension | With inflammation hemorrhage or neoplasm involving the brain or the meninges Look for papilledema and consult neurosurgeon before performance of lumbar puncture |
| Reflex irritation | With peritonitis pancreatitis gastric crises in tabes dorsalis and lead colic |
| | In absence of acute toxemia and inflammation, look for basophilic stippling of erythrocytes in plumbism |
| | Perform serologic test and gold curve for tabes (p 3737) |
| | With acute symptoms consult surgeon for abdominal puncture preparatory to laparotomy (p 1920) |
| Generalized Protusion of the Abdominal Wall | |
| In Infancy | With cretinism rickets achondroplasia and mongolism (p 2730) |

| | |
|--|--|
| In the Adult | <p>Gastrovisceroptosis lumbar lordosis postoperative ventral hernia diastasis recti paralysis of muscles of anterior abdominal wall pregnancy obesity ascites congenital megacolon, splenomegaly (p 1129) hepatomegaly (p 1973) and large abdominal tumors (p 1750)</p> <p>Test muscle strength in supine and erect positions Examine uterus in suspected gravidity Look for fluid wave and perform exploratory abdominal puncture in ascites (p 1921) Do barium meal and barium enema for demonstration of gastrovisceroptosis or congenital megacolon (p 1871)</p> |
| Localized Protrusion of the Abdominal Wall | Ventral, epigastric umbilical incisional inguinal and femoral hernias Examine supine and erect after cough |
| Visible Peristalsis | |
| Epigastric | In infancy with congenital pyloric stenosis (p 2730) History of persistent vomiting with palpable tumor |
| Generalized | With hyperperistalsis in lean and cachectic patients Intestinal or pyloric obstruction with history of persistent vomiting and obstipation (p 1873) |
| | Get radiograph to distinguish dilated and collapsed loops of bowel (p 1874) Prepare for intubation (p 1823) and laparotomy (p 1820) |
| Tumors | Desmoids of rectus Hematoma of rectus sheath with sudden onset following persistent vomiting or cough |
| Dilatation of Superficial Veins | Portal obstruction due to hepatic cirrhosis (caput medusae) Thrombosis or stenosis of inferior vena cava |
| Abnormalities of Superficial Reflexes | |
| Hyperactivity | In the ticklish |
| Inequality or Inconsistency | Early sign of poliomyelitis (p 457) In presence of stiffness of spine and fever get cerebrospinal fluid (p 3735) |
| Absence | With increased intracranial pressure meningitis general paresis tabes dorsalis multiple sclerosis and lateral sclerosis |
| | Make complete examination of cerebrospinal fluid (p 3735) Examine fundus for papilledema with increased tension, and for atrophy in paresis and multiple sclerosis Get neurologic status for confirmatory findings |
| Umbilical Discharge | Omphalitis in infancy Persistence of omphalo-mesenteric vessels omphalo-mesenteric duct or Meckel's diverticulum with chronic or recurrent inflammation (p 1864) |
| Localized Infection: Fistula or Sinus | Infected abdominal wound Retained suture Pointing from abscess of peritoneum, pelvis or spine Communication with hollow bowel (fecal fistula) |
| | Visualize with lipiodol and consult surgeon Make biopsy for evidences of tuberculosis or actinomycosis (p 489) |

THE ABDOMINAL CAVITY AND ITS CONTENTS

As with the contents of the thoracic cavity the examination of intra-abdominal structures must be made inferentially. Inspection, percussion and auscultation furnish data of lesser significance. The greatest amount of information is gained through careful *palpation*.

The aims of abdominal examination are (1) to examine the *individual organs* with respect to their position, size, form, consistency, mobility and tenderness (2) to *detect tumors* (3) to determine the presence of *excess fluid* in the abdominal cavity (4) to note *tender areas* and *spasm* of the abdominal wall.

THE ZONES OF THE ABDOMEN

The abdomen is arbitrarily divided into anatomical zones. The terms are self-explanatory. They include the *right and left hypochondriac* areas, the *epigastrium*, the *umbilical* and *right and left lumbar* regions, the *hypogastric* zone and the two *iliac fossae*.

ANATOMIC REVIEW

The Solid Viscera—The solid abdominal viscera occupy relatively constant positions. The *spleen* is in the left and the *liver* in the right hypochondrium. The *kidneys* in the respective lumbar regions posteriorly. The right kidney lies near the lower edge of the liver while the left kidney lies posterior to and below the spleen.

The Liver and Bile Passages—The liver, the largest gland in the body, is situated in the upper and right part of the abdominal cavity. It occupies almost the whole of the right hypochondrium, the greater part of the epigastrium and extends into the left hypochondrium as far as the midclavicular line. The organ is reddish brown and may weigh 1200 to 1800 gm.

The liver has five *surfaces*, four of which—the *superior*, *anterior*, *posterior* and *right lateral*—are in proximity to the parietes or the diaphragm. The *inferior* or *visceral* surface has a transverse cleft which contains the openings of the portal vein, the hepatic artery and the common bile duct. Between the quadrate and the right lobe there is situated the pear-shaped gallbladder.

The liver receives its blood supply from the hepatic artery and the portal vein. The *hepatic artery* is a branch of the celiac axis. It nourishes the vessels, biliary ducts and interlobular connective tissue. The *portal vein*, formed by the junction of the splenic and superior mesenteric vessels, is the nutrient vessel of the parenchymal tissue. It breaks down into a system of capillaries which later unite and eventually open into the inferior vena cava. Thus the portal blood has passed through two sets of capillaries before returning to the heart. See *Differential Diagnosis of Enlargement of the Liver* (p. 1973).

The *hepatic duct* is formed from the subsidiary bile channels. It passes downward and to the right for about $1\frac{1}{2}$ to 2 inches. Between the layers of the lesser omentum it is joined at an acute angle by the cystic duct of the gallbladder and the two form the common bile duct which opens into the duodenum.

The *gallbladder* serves as a reservoir for bile. It lodges in a fossa on the under surface of the right lobe of the liver. It is approximately 3 to 4 inches in length and 1 inch in breadth at its widest part. It normally holds 30 to 40 cc.

The gallbladder terminates in the *cystic duct*, about $1\frac{1}{4}$ inches long. This duct passes obliquely downward and to the left from the neck of the gallbladder and joins the hepatic duct. The mucous membrane is thrown into a series of crescentic folds which act as valves.

The *common bile duct* is about 3 inches long and approximately of the diameter of a goose quill. It descends along the right border of the lesser omentum behind the first portion of the duodenum. It then passes between the head of the pancreas and the descending portion of the duodenum and for a short distance occupies a position along the right side of the terminal portion of the pancreatic duct, with which it passes obliquely through the wall of the descending portion of the duodenum. The two ducts unite in the *ampulla* of Vater, though independent openings persist in perhaps 10 per cent of cases. Circular muscle fibers constituting the so-called *sphincter of Oddi* are continuous with the longitudinal fibers of the duct and surround the termination of the duct in the ampulla. See *Differential*

Diagnosis of Jaundice (p 1931) *Jaundice in the Newborn* (p 261) *Obstructive Jaundice* (p 1934) *Swellings and Tumors in Right Upper Quadrant* (p 1937) *Pain in Right Upper Quadrant* (p 1939)

The Kidneys—See p 2207

The Spleen—The spleen is situated principally in the posterior portion of the left hypochondrium. It is obliquely placed its long axis following the course of the tenth rib. It lies between the fundus of the stomach and the diaphragm. It usually measures about 5 inches in length. The normal spleen weighs 160 to 210 gm. Supernumerary or accessory spleens are frequently found in the neighborhood of the main organ.

The external or phrenic surface of the spleen lies in close proximity to the under surface of the diaphragm. The inner surface reveals an anterior or gastric and a posterior or renal portion. These surfaces are divided by the hilum which is provided with apertures for the vessels and nerves.

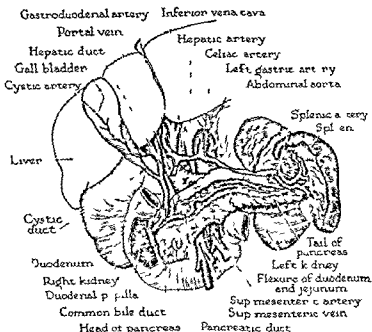


Fig 1032.—Relative positions of liver spleen pancreas gallbladder and duodenum

The splenic artery is a branch of the celiac axis which in turn is derived from the abdominal aorta. The splenic vein is a portion of the portal system uniting with the superior mesenteric vessels to form the portal vein. In consequence the returning blood from the splenic parenchyma traverses the spleen and then the portal capillaries before entering the right auricle. See *Definitive Diagnosis of Splenomegaly* (p 1129).

The Hollow Viscera: The Esophagus—The esophagus or gullet is a muscular membranous canal approximately 10 inches in length extending from the oropharynx to the stomach. The esophagus begins at the upper border of the cricoid cartilage opposite the sixth cervical vertebra. It descends along the front of the vertebral column through the posterior mediastinum and passes through the diaphragm entering the abdomen at a point opposite the eleventh thoracic vertebra about 1 inch to the left of the median line. The terminal inch of the esophagus occupies a position within the abdominal cavity. In its course through the neck thorax and abdomen the esophagus is in intimate anatomical relationship with the trachea and bronchus, the common carotid artery, the descending aorta, the thoracic duct,

the thoracic sympathetic chain the lungs and pleurae the diaphragm the inferior vena cava and the posterior surface of the pericardium and heart

The lumen of the esophagus has *three main points of narrowing* (1) at the upper end where the cricopharyngeus muscle forms a sphincter (2) at the level where the left bronchus crosses the gullet and (3) at the site of the passage of the esophagus through the diaphragm and into the abdominal cavity

The *mucosa* of the esophagus is formed by stratified squamous epithelium In the terminal portion there are often located islets of gastric mucous membrane composed mostly of mucus secreting cells but occasionally consisting of acid secreting parietal cells The mucous membrane of the esophagus is not very sensitive to ordinary chemical or physical stimulation Occasionally a violent irritation gives rise to pain felt in the epigastrium and radiating substernally to the neck In ordinary digestion food is constantly being regurgitated into the lower end of the esophagus Despite this no sensation is registered in normal individuals The pharyngeal end of the tube however may be sensitive as shown by the common experience of regurgitating water brash

The *wall* of the esophagus has an inner circular and an outer longitudinal layer of muscle In the upper two thirds this muscle is striated while in the lower third it is smooth A small percentage of normal individuals have a striated muscle tube throughout the extent of the gullet

The *arterial blood supply* of the esophagus in its upper portion is derived from branches of the carotid arteries In the thorax the arterial blood supply arises from the aorta and the intercostal arteries The lowermost portion derives its arterial blood from the left gastric artery

In general the *venous return* drains into veins which accompany the arteries In the lower third of the esophagus there is a very complex venous plexus in the submucosal and muscular layers This plexus continuous with a similar one lying in the submucosa of the stomach at the cardia, drains through esophageal veins into the azygos and hemiazygos veins, thence into the superior vena cava As these veins have no valves in the vertical position of the body there is an unsupported column of blood extending from the superior vena cava to the level of the lowermost portion of the esophagus

The *lymphatics* of the esophagus drain into the deep cervical nodes and the nodes of the posterior mediastinum The cervical glands are often involved in metastatic deposits from carcinoma of the esophagus

The esophagus is supplied by cholinergic and adrenergic systems It is doubtful whether the latter has any motor control over the gullet whose normal activity is largely dependent upon the integrity of the cholinergic or vagal fibers

See *Differential Diagnosis of Dysphagia and Pain on Swallowing* (p 173) *Hematemesis* (p 1764)

The Stomach—The stomach is a mucomuscular sac which serves as an organ of digestion and as a temporary receptacle for food It varies greatly in size shape and position The normal stomach is described as being situated in the epigastric and left hypochondriac regions of the abdomen under which circumstances it is of the *steer horn* type These conditions prevail in the stocky type of individual (p 3489) Patients of the *ptotic habitus* (p 3488) rarely present the normal as described in textbooks Most often the stomach is of the *"fish hook"* variety Its body is situated for the most part in the umbilical or even the hypogastric zone

Whether the stomach is of the *steer horn* or *fish hook* type two borders two surfaces and several anatomical subdivisions are recognizable The upper border is the *shorter or lesser curvature* which lies beneath the lower surface of the liver to which it is attached by the lesser omentum The *lower or greater curvature* faces the pelvic portion of the abdominal cavity From it is suspended the *omentum* which covers the abdominal contents in the manner of an apron

The stomach has *anterior* and *posterior surfaces* The *cardia* is that portion closest to the entrance of the esophagus The *fundus* is the superior part of the stomach, along the left border which occupies a position higher than the esophageal opening into the cardia The major portion of the stomach is the body or *corpus* which extends from the cardia and fundus to the *incisura angularis* a wedge-shaped indentation on the lesser curvature The terminal portion of the stomach is the *antrum* which extends from the incisura angularis to the *pylorus* The antral portion of the stomach is of the greatest clinical significance since it is here that organic lesions are most apt to occur

The *mucosa* of the stomach contains several different types of cells The *zymogenic cells* secrete pepsinogen the precursor of *pepsin* The *parietal cells* manufacture *hydrochloric acid*

chloric acid There are in addition *mucus secreting cells* The parietal and zymogenic cells are mainly located along the lesser curvature the anterior and posterior walls near the cardia, and in the upper portion of the body of the stomach The zymogenic cells also extend to the greater curvature and are found in the fundus Parietal cells are likewise present in smaller numbers in the fundus but are infrequently seen in the antrum The mucus-secreting cells are scattered throughout the stomach and form the majority of the cells in the antrum and cardia

Small *islands of intestinal mucosa* are also found scattered throughout the stomach These occur mainly in the antrum and especially in the region close to the pylorus They

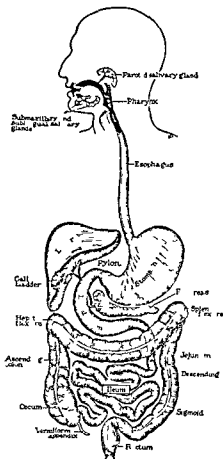


Fig 1033—Diagram of the organs of the digestive system

are considered variously as a congenital anomaly or the result of recurrent inflammatory disease producing heterotopic replacement.

Beneath the mucosa of the stomach is the *muscular layer* consisting of circular oblique and longitudinal smooth muscle fibers with a recognizable sphincteric formation of the circular layer at the pylorus

The outermost or *serosal layer* of the stomach consists of peritoneum and a subperitoneal areolar tissue At the lesser curvature the peritoneum stretches to form the *gastro-hepatic ligament* At the greater curvature the omentum spreads over the abdominal

viscera The greater curvature is also attached by the *gastrocolic* and *gastrosplenic ligaments* to the colon and spleen respectively

The arterial blood supply of the stomach arises from the celiac axis by two large branches which run along the lesser curvature of the stomach The *left gastric artery* is derived from the celiac itself the *right gastric artery* is a branch of the hepatic artery The greater curvature has an additional arterial supply through the right and left *gastroepiploic arteries* The former is a branch of the superior pancreaticoduodenal while the left is derived from the splenic artery Additionally a variable number of branches from the splenic artery run from the anterior and posterior walls of the stomach in the region of the fundus These vessels as well as the gastric branches of the left gastric artery anastomose with the arterial blood supply of the lower end of the esophagus

The *venous return* follows the arterial pattern and drains almost entirely into the portal veins The *lymphatics* follow the course of the arteries to lymph nodes along the lesser curvature especially near the pylorus It is here that metastases are sought in malignancies of the organ

The stomach is innervated by the vagal and sympathetic trunks The *right and left vagi* continue downward from the thorax and supply the anterior and posterior walls of the stomach These great nerve trunks terminate in the muscle fibers and communicate with postganglionic nerves which penetrate into the mucosa The *cholinergic system* is the principal regulatory mechanism of secretory and motor functions *Adrenergic fibers* are derived from the thoracic sympathetic chain with ganglionic connections in the celiac plexus From here they follow the blood vessels into the gastric tissues proper Adrenergic fibers at most are of minor importance in relation to the functions of the stomach

Duodenum—The duodenum is the shortest, widest and most fixed portion of the small intestine It has no mesentery and is only partially invested by peritoneum The duodenum begins at the pylorus and passes backward upward and to the right The *first portion (duodenal bulb or cap)* is attached to the lesser omentum It is intra abdominal but extra peritoneal The *second portion* of the duodenum lies parallel to the vertebral column Its upper portion is situated over the lower pole of the right kidney Its left border is adjacent to the head of the pancreas

The *common bile duct* empties into the second portion of the duodenum after having been joined by the *major pancreatic duct* Both of these structures open into the papilla of Vater which lies on the mesial border of the second portion of the duodenum Not infrequently there is an *accessory pancreatic duct* opening separately in the close vicinity The *third portion* of the duodenum is directed almost horizontally to the left in its first half After crossing the vertebral column it turns proximally to the region of the *ligament of Treitz* which may be the site of herniations and obstruction In the region of the second lumbar vertebra the duodenum joins the jejunum forming the acute *duodenojejunal flexure*

The duodenum lies in close proximity to the liver and gallbladder the right kidney and its vessels the pancreas the aorta the inferior vena cava the portal vessels the bile and pancreatic ducts the stomach colon and small intestines and the thoracic duct and the retroperitoneal spaces

The *arterial supply* of the duodenum is by way of branches from the hepatic artery and the superior mesenteric The *veins* drain into the portal system the *lymphatics* into the thoracic duct

Innervation of the duodenum occurs through filaments from the solar plexus There are direct branches from the vagus to the intrinsic duodenal ganglia Adrenergic fibers arise from the abdominal sympathetics

The *duodenal mucous membrane* is characterized by numerous villi thrown into longitudinal folds These villi are richly supplied with blood vessels lymphatics and numerous secreting glands

See *Differential Diagnosis of Hematemesis* (p 1761) *Dyspepsia* (p 170) *Polyphagia* (p 1776) *Anorexia* (p 1779) *Epigastric Pain* (p 1788) *Epigastric Swellings and Tumors* (p 1814)

The *Small and Large Intestines Including the Vermiform Appendix* JEJUNUM AND ILEUM—The small intestine is a coiled tube 20 to 22 feet long The first 12 inches the duodenum have been described At the *duodenojejunal juncture* the small bowel acquires its mesentery and becomes intraperitoneal The jejunum forming the proximal portion of the small bowel lies in the upper and left portion of the abdominal cavity The ileum, shorter in length and smaller in diameter occupies a position in the lower and right portion

of the abdominal cavity. The ileum opens into the cecum by the means of a fold of tissue called the *ileocecocolic valve* which is usually completely competent.

The intestinal secreting surface is greatly increased by numerous transverse plaques and folds. The surface is covered with numerous villi richly supplied with blood and lymph functioning for the absorption and digestion of food.

COLON—The colon begins at the ileocecal valve. It consists of a lower blind end the cecum with its attached vermiform appendix, an ascending portion, the hepatic flexure, the transverse colon, the splenic flexure, the descending colon, the sigmoid flexure, and the rectum.

Most of the colon occupies a fairly fixed position within the abdominal cavity, though there may be developmental variations. Ordinarily the cecum is in the right lower quadrant. The hepatic and splenic flexures are relatively fixed, respectively in the right and left upper quadrants. The ascending colon occupies the right lumbar gutter, the descending colon the left. The transverse colon has a variable position according to the normal descriptions; it traverses the epigastrium. More often than not it hangs down like a hammock until in the extreme instances of *colopropsis* (p. 1808) it actually assumes a pelvic position.

The external surfaces of the colon are distinguished from the loops of small bowel by *taeniae* and prominent longitudinal muscle bands (*taeniae*) on the free surfaces.

MUCOSA—The mucous membrane of the small intestine has a variety of cellular constituents. In the duodenal portion there are the glands of Brunner located in the mucosal as well as the submucosal layers. These empty into the small intestine by way of tiny ducts located at the bases of the tubular glands of the duodenum. The mucous glands and epithelial cells secrete a variety of enzymes which participate in the final breakdown of food products. In addition there are cells whose functions are not known. These include the *Paneth* and *enterochromaffin cells*.

Occasionally the small bowel contains ectopic foci of gastric mucous membrane containing gastric secreting cells. They occur especially in the Meckel's diverticulum (p. 1804) usually located about 2 feet from the ileocecal valve. *Aberrant pancreatic tissue* has also been found distributed throughout the entire small intestine but most especially in its duodenal portion.

MUSCULARIS—Beneath the mucosa, as in the stomach, there is the muscularis mucosae which functions independently of the muscle coat. It serves to give a wide variety of pattern formations to the mucous membrane so that food is brought into more intimate contact with the secreting surface.

Beneath the muscularis mucosae are the inner circular and outer longitudinal muscles. In the colon the external longitudinal muscle is divided into three bands called *taeniae*, one of which runs along the anterior surface of the colon taking its origin in the cecum at the root of the appendix. The other *taeniae* are distributed laterally on the walls of the bowel.

VASCULAR SUPPLY—The vessels, nerves and lymphatics of the gut enter and emerge at the mesenteric border. The small bowel and the right half of the colon receive branches from the superior mesenteric artery. These vessels divide and subdivide in an arcuate fashion supplying the intestinal wall. The left half of the colon derives its blood supply from the inferior mesenteric by means of the left colic, the superior hemorrhoidal and the sigmoid arteries. The veins of the bowel drain into the mesenterics which unite with the splenic to form the portal system. Thus the products absorbed from the gut are carried in the entirety to the liver.

The mucosa of the small intestine is marked by longitudinally arranged oval deposits of lymphoid tissue (*Peyer's patches*). Lymphatic plexuses are situated in the villi of the large bowel. They are known as *lacteals* because of the milky appearance. They function to absorb fatty substances from the intestine. The lacteals unite to form intestinal lymphatic trunks which drain into the receptaculum chyli and thence into the thoracic duct. Additionally there are lymphatic glands in the mesentery.

NERVE SUPPLY—The small intestine receives nerve filament from the *cholinegic* and *adrenergic* systems. All fibers emanate from the solar plexus which surrounds the celiac artery and its branches. In the wall of the small intestine these filaments are brought into relation with the plexuses of Auerbach and Meissner from which fibers are distributed to the muscles and mucous membranes where they serve for motor and secretory functions. As in the stomach the greater control is exercised through the cholinergic system. The sympathetic outflow supply follows the plantar nerve whose ganglionic in the thoracic sympathetic chain. The distal colon derives its cholinergic innervation from the sacral outflow.

THE CECUM AND VERMIFORM APPENDIX—The cecum is the first portion of the colon and occupies a position below the transverse ileum passing just above the ileocecal valve. It is 2 to 3

viscera The greater curvature is also attached by the *gastrocolic* and *gastrosplenic ligaments* to the colon and spleen respectively

The arterial blood supply of the stomach arises from the celiac axis by two large branches which run along the lesser curvature of the stomach The *left gastric artery* is derived from the celiac itself the *right gastric artery* is a branch of the hepatic artery The greater curvature has an additional arterial supply through the right and left *gastroepiploic arteries* The former is a branch of the superior pancreaticoduodenal while the left is derived from the splenic artery Additionally a variable number of branches from the splenic artery run from the anterior and posterior walls of the stomach in the region of the fundus These vessels as well as the gastric branches of the left gastric artery anastomose with the arterial blood supply of the lower end of the esophagus

The *venous return* follows the arterial pattern and drains almost entirely into the portal veins The *lymphatics* follow the course of the arteries to lymph nodes along the lesser curvature especially near the pylorus It is here that metastases are sought in malignancies of the organ

The stomach is innervated by the vagal and sympathetic trunks The *right and left vagi* continue downward from the thorax and supply the anterior and posterior walls of the stomach These great nerve trunks terminate in the muscle fibers and communicate with postganglionic nerves which penetrate into the mucosa The *cholinergic system* is the principal regulatory mechanism of secretory and motor functions *Adrenergic fibers* are derived from the thoracic sympathetic chain with ganglionic connections in the celiac plexus From here they follow the blood vessels into the gastric tissues proper Adrenergic fibers, at most are of minor importance in relation to the functions of the stomach

Duodenum—The duodenum is the shortest widest and most fixed portion of the small intestine It has no mesentery and is only partially invested by peritoneum The duodenum begins at the pylorus and passes backward upward and to the right The *first portion (duodenal bulb or cap)* is attached to the lesser omentum It is intra abdominal but extra peritoneal The *second portion* of the duodenum lies parallel to the vertebral column Its upper portion is situated over the lower pole of the right kidney Its left border is adjacent to the head of the pancreas

The *common bile duct* empties into the second portion of the duodenum after having been joined by the *major pancreatic duct* Both of these structures open into the *papilla of Vater* which lies on the mesial border of the second portion of the duodenum Not infrequently there is an *accessory pancreatic duct* opening separately in the close vicinity The *third portion* of the duodenum is directed almost horizontally to the left in its first half After crossing the vertebral column it turns proximally to the region of the *L4 segment of Treitz* which may be the site of herniations and obstruction In the region of the second lumbar vertebra the duodenum joins the jejunum forming the acute *duodenojejunal flexure*

The duodenum lies in close proximity to the liver and gallbladder the right kidney and its vessels the pancreas the aorta the inferior vena cava the portal vessels the bile and pancreatic ducts the stomach colon and small intestines and the thoracic duct and the retroperitoneal spaces

The *arterial supply* of the duodenum is by way of branches from the hepatic artery and the superior mesenteric The *veins* drain into the portal system the *lymphatics* into the thoracic duct

Innervation of the duodenum occurs through filaments from the solar plexus There are direct branches from the vagus to the intrinsic duodenal ganglia Adrenergic fibers arise from the abdominal sympathetics

The *duodenal mucous membrane* is characterized by numerous villi thrown into longitudinal folds These villi are richly supplied with blood vessels lymphatics and numerous secreting glands

See *Differential Diagnosis of Hematemesis* (p 1761) *Dyspepsia* (p 1710) *Polyphagia* (p 176) *Anorexia* (p 1779) *Epigastric Pain* (p 1788) *Epigastric Swellings and Tumors* (p 1814)

The Small and Large Intestines Including the Vermiform Appendix **JEJUNUM AND ILEUM**—The small intestine is a coiled tube 20 to 22 feet long The first 12 inches the *duodenum* have been described At the *duodenojejunal juncture* the small bowel acquires its mesentery and becomes intraperitoneal The *jejunum* forming the proximal portion of the small bowel lies in the upper and left portion of the abdominal cavity The ileum, shorter in length and smaller in diameter occupies a position in the lower and right portion

See *Differential Diagnosis of Generalized Abdominal Pain* (p 1748) *Involuntary Rigidity of the Abdominal Wall* (p 1746) *Generalized Abdominal Swellings due to Tumors* (p 150) *Darrhea* (p 1840) *Melena and Tarry Stools* (p 1843) *Constipation* (p 1852) *Pain in Right Lower Quadrant* (p 1880) *Pain in Left Lower Quadrant* (p 1866) *Swellings and Tumors of Right Lower Quadrant* (p 1886) and *Left Lower Quadrant* (p 1870) *Pain in Umbilical Region* (p 1887)

THE ANUS—The normal anus appears as a puckered orifice. The circumanal skin is usually pigmented, the degree being increased in patients who habitually take castors. The anal canal is $1\frac{1}{4}$ inches in length. It extends from the lower end of the rectum to the anus. The junction with the rectum is marked by the *mucocutaneous* or *pectinate line*. Here the narrow anal canal gives into the wider lumen of the rectum which in its terminal 2 or 3 inches is characterized by longitudinal folds forming the *columns of Morgagni*. These columns are very vascular and become the site of the pathological condition of *internal hemorrhoids* (p 1916).

At the bases of the Morgagni columns appear the *valves and crypts of Morgagni*. The latter are vulnerable to infection giving rise to *cryptitis* which often goes on to formation of *fissures* and *perineal fistulae*. Additionally there are small *papillae* in close proximity to the bases of the columns of Morgagni or on the folds of the anal valves. These become inflamed and hypertrophied giving rise to a local *papillitis*, a frequent cause of sphincteric spasm and pain.

The anal canal is surrounded by an *external* or *voluntary* and *internal* or *involuntary sphincteric muscle*. An almost imperceptible circular depression known as the *white line of Hilton* (the *intersphincteric line*) marks the demarcation between the two muscles.

The anal canal is well supplied with *sensory nerves*. It has poor blood supply. The *arterial vessels* come from the hemorrhoidal and middle sacral branches. The *venous drainage* of greater clinical importance is divided into two parts. The lower represented by the middle or inferior hemorrhoidal veins arises below the mucocutaneous line around the anal canal. These drain into the *caval circulation*. The superior hemorrhoidal veins arise just above the mucocutaneous junction and empty into the *portal circulation*. The hemorrhoidal veins have no valves. Back pressure and increased intra abdominal pressure are readily transmitted giving rise to the production of hemorrhoids (p 1916).

See *Differential Diagnosis of Ano-perineal Pain* (p 1913) *Pruritus Ani* (p 1916) *Incontinence of Feces* (p 1915)

PALPATION OF THE ABDOMINAL VISCERA

THE HOLLOW VISCERA

Normally the hollow abdominal viscera are not palpable but it is not at all uncommon to feel the *cecum* and *sigmoid flexures* when they are spastic filled with feces or distended with gas—findings which must be differentiated from a true abdominal neoplasm.

Additionally the *dilated stomach* or the *distended urinary bladder* may be felt. These disturbances should always be sought in postoperative and toxic patients. The *enlarged or gravid uterus* may become readily palpable when sufficiently increased in size to rise above the symphysis.

THE SOLID ABDOMINAL VISCERA

Under normal circumstances the solid abdominal viscera are not palpable except perhaps for the edge of the *liver* or the tip of the *right kidney*. In thin individuals the *abdominal aorta* is readily recognized. It pulsates and may be tender. It is not to be confused with an aneurysm (p 1026) which is a rare clinical condition.

The Special Technics of Abdominal Palpation for the Solid Viscera—To palpate the solid viscera the right handed examiner stands or preferably sits comfortably at the right side of the patient. Special efforts are made to palpate the various organs. Since the resistance of the abdominal wall is

inches long and slightly wider and in its normal position lies upon the right iliopsoas muscle. As a result of developmental anomalies the cecum may lie on the right kidney below the liver. In elderly and ptotic patients it may descend and constitute a pelvic cecum. The cavity of the cecum is continuous with the ascending colon above and the appendix below. It is separated from the ileum by the interposition of the ileocecal valve.

The vermiform appendix attaches to the posteromesial border of the cecum. It can always be located by tracing distally the anterior longitudinal band. Like the cecum it may be situated in almost any portion of the abdominal cavity but its usual positions are the right lower quadrant, right upper quadrant and beneath the brim of the pelvis. The appendix is

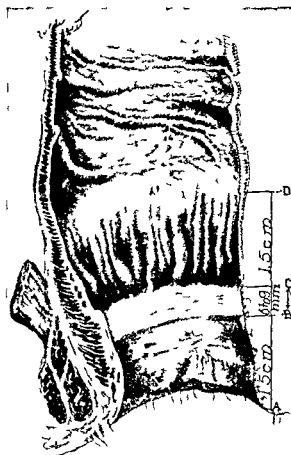


Fig. 1034.—Divisions and landmarks of the anal canal viewed in frontal section. A The anocutaneous line (anal verge) B-C Pecten band C Pectinate (dentate) line D Anorectal line*.

usually cylindrical and from 2 to 4 inches long. Its coats are similar to those of the rest of the colon but the muscularis is poorly developed and may be deficient. The lumen is of variable size and may be irregular or obliterated. Its orifice into the cecum is usually guarded by a crescentic fold which acts as a valve obstructing the entrance to fecal material. Incompetence of this valve may be a cause for the frequency of acute appendicitis.

The relationship of appendix to cecum is variable accounting for the bizarre symptom complexes seen in acute inflammation. The appendage usually crosses the psoas muscle and hangs down freely over the pelvic brim. It may however be retrocecal, retroperitoneal, pelvic and attached to the urinary bladder or internal genitalia of the female or situated so high up as to be virtually subhepatic.

the female breast the prophylaxis of malignancy is best accomplished by these disciplines

See *Differential Diagnosis of Generalized Abdominal Swellings due to Solid Tumors* (p 1750) *Swellings and Tumors in Left Upper Quadrant* (p 1849) *Left Lower Quadrant* (p 1870) *Right Lower Quadrant* (p 1886) *Right Upper Quadrant* (p 1957) *Hepatomegaly* (p 1973) *Splenomegaly* (p 1129) *Enlargement of the Kidney* (p 2230) *Swellings and Tumors in the Hypogastrium* (p 2621)

PERCUSSION OF THE ABDOMEN

Percussion of the abdominal cavity may yield considerable information. Varying degrees of *tympany* are present depending upon the relative gaseous content of the hollow viscera.

Liver Dulness—Normally there should be dulness in the right upper quadrant corresponding to the position of the liver. *Decrease or obliteration of liver dulness* is a physical sign of great importance indicating that there is free gas in the abdominal cavity or an appreciable shrinkage in the size of the liver.

Shifting Dulness—Shifting dulness in the flanks may be produced by fluid feces in the large bowel. Usually it indicates the presence of ascitic fluid in the peritoneal cavity. To elicit the presence of shifting dulness the flank is percussed with the patient lying in one and then the other lateral decubitus. The upper flank gives a tympanitic note replaced by flatness when the patient shifts position.

Fluid Wave—Corroborating the percussive evidence of free fluid in the peritoneal cavity are the palpatory data obtained by eliciting the fluid wave. The patient presses the thenar side of his hand along the area from the xiphoid to symphysis. The examiner places the palms of his hands laterally and taps against the abdominal wall with the finger of one hand. In the presence of free fluid the impulse of the wave is felt by the stationary hand. This sign is not invariably present even when there are large amounts of free fluid in the abdominal cavity.

See *Differential Diagnosis of Tympanites* (p 1878) and *Ascites* (p 1921).

Hypogastric Dulness—Hypogastric dulness or flatness is commonly due to *retention of urine* (p 2264) and/or *enlargement of the uterus* (p 2254). When in doubt the patient should be catheterized and the percussion repeated.

Epigastric Dulness—The routine examination of patients following operation or of those with chronic disease should always include percussion of the epigastrium. This will insure early detection of a dilated or *distended stomach* (p 1769).

Splenic Dulness—Percussion of the left upper quadrant for splenic enlargement is too unreliable to warrant definite inferences.

AUSCULTATION OF THE ABDOMEN

Abdominal auscultation is a form of examination that is frequently neglected. Normally there may be heard gurgling sounds (*borborygmi*) produced as the result of peristaltic waves traversing an area of gut which

increased with inspiration and decreased with expiration the latter is the logical time for deep palpation. During inspiration the hand should rest quietly on the abdominal wall, while in expiration it should penetrate more deeply in the effort to feel the structure.

The patient lies flat without a pillow and breathes with the mouth open in order to obtain the greatest amount of abdominal relaxation. Flexion of the thighs on the trunk increases the degree of relaxation. It is wise to begin with light palpation over nontender areas and resort to deeper palpation in areas of suspected disease when the patient is at ease. Some patients have difficulty in breathing and relaxing the abdominal wall when their attention is focused on the procedure. Instructing such a patient to sigh is frequently helpful.

The Liver and Gallbladder—These organs are best examined by placing the left hand under the patient's lower ribs and palpating the region of the costal border with the fingers of the right hand. During inspiration particularly if the patient is lifted almost bodily by the left hand it is possible to feel the edge of the normal liver. The liver should not be tender.

The gallbladder is not palpable in health and there should not be tenderness in the region of the normal gallbladder which usually is situated at the end of the tenth rib in the midclavicular line (Murphy's point).

The Kidneys—Before palpating the kidneys inspection of the lumbar region may reveal a visible swelling. Thus a *perirenal abscess* (p. 2560) may cause a protrusion in the loin sometimes with edema of the overlying skin.

Palpation of the kidney region is carried out bimanually. In the usual position the patient lies on the back with the knees flexed. To palpate the right kidney, the right hand of the examiner is placed anteriorly and the left posteriorly. For palpating the left kidney the position of the hands is reversed. The patient is asked to breathe in and out through his mouth while the examiner gradually presses his hands gently together. An occasional firm tap with the posterior hand while the patient stops breathing in the expiratory phase permits *ballottement* of the kidney. This maneuver may reveal a ptosed kidney or a kidney mass not otherwise palpable.

The position of the patient on the back is known as the *Guyon position* in contradistinction to the *position of Israel* in which the patient lies on the side with legs and thighs semiflexed. The latter position sometimes reveals a palpable kidney or a mass associated with a kidney. Palpating the kidney in the erect posture is of value in detecting *nephroptosis* (p. 2293). Normally the lower pole of the right kidney is more readily palpable than the left.

The Spleen—For the palpation of the spleen the left hand of the examiner arches over the body and is placed posteriorly beneath the lower ribs. The fingers of the right hand rest below the left costal border. With *superficial palpation* an attempt is made to feel the lower border of the viscus while the patient breathes deeply. The maneuver is repeated with the patient in the right lateral decubitus. The tip of the spleen may be felt in health in a particularly thin patient. Any greater enlargement requires investigation.

Pelvic Organs—Only by firm adherence to the rule of including in the physical examination rectal and vaginal investigation (p. 3648) can the practitioner avoid grave errors of omission. As with re . of

CHAPTER 161

PHYSICAL EXAMINATION LOCOMOTOR AND SKELETAL SYSTEMS, PERIPHERAL VESSELS NERVOUS SYSTEMS

THE LOCOMOTOR AND SKELETAL SYSTEMS

A GROSS survey of the *muscles bones and articulations* of the trunk and extremities is conducted by the practitioner. More intensive investigation is the province of the specialist orthopedic surgeon.

THE TRUNK AND BONY PELVIS ANATOMIC REVIEW

The vertebral column is a flexible rod sustaining the trunk and head. It is set firmly into the pelvis and is balanced or guyed by means of ligaments and muscles. The chief muscle groups are (1) the *erector spinae* (2) the *abdominals* (3) the *glutei* (4) the *psoas* and (5) the *hamstrings*. The back muscles are covered by an extensive lumbodorsal fascia. The trunk is balanced upon the femora by the action of the muscles controlling the hips. The succeeding joints are similarly sustained by their intrinsic musculature.

The summation of these dynamic forces determines the posture of the individual.

The Vertebral Column.—The vertebral column is divided into *cervical thoracic lumbar sacral* and *coccygeal portions*. It contains 33 vertebrae: 7 cervical 12 thoracic 5 lumbar 5 sacral (fused) and 4 coccygeal (fused).

The Curves of the Vertebral Column.—Viewed laterally the vertebral column presents several normal curves. The *cervical curve* is convex forward. It extends from the apex of the odontoid process to the middle of the second thoracic vertebra. The *thoracic curve* is concave forward and extends to the twelfth thoracic vertebra. The *lumbar curve* is convex anteriorly and extends to the first sacral vertebra where the *pelvic curve* which is concave anteriorly begins. This is continued down to the tip of the coccyx.

These curves are not constant. They do not exist in the newborn infant whose spine has the shape of a C with a concavity anteriorly from occiput to coccyx. The cervical curve develops when the infant begins to hold up his head; the lumbar curve when he sits and stands. The curves tend to become exaggerated with advancing age. There are likewise individual variations in the degree of these curves. Some of these are consistent with good posture. A slight degree of lateral curvature may be physiological. This is usually convex to the right in the dorsal region. It is attributed to the greater use of the right upper extremity.

The Vertebral Articulations.—Each vertebral body articulates with the one above and below through the corresponding articular surfaces. The adjacent surfaces are separated by an *intervertebral disk*. Each disk contains a jelly like *nucleus pulposus* which is held under pressure within a peripheral *fibr cartilaginous ring*. The vertebral body rocks over this disk as over a ball bearing.

The Vertebral Ligaments.—In front of the vertebral bodies runs the very strong *anterior longitudinal ligament*. The *posterior longitudinal ligament* is much weaker especially at the lumbosacral junction. This is the usual site for protrusion of the nucleus pulposus (p. 304). From the inner portion outward the vertebral arches are joined by: (1) the *ligamenta flava* which connect the laminae of the adjacent vertebrae (2) the *capsular ligaments* that contain the *superior articular process* of the vertebra below and the *inferior articular process* of the vertebra above (3) the *interspinous ligaments* connecting the adjoining spinous processes (4) the *transverse ligaments* between the transverse processes and (5) the *supraspinous ligaments* from the tips of the spinous processes.

Subluxation of the sacral processes without actual dislocation may be possible if the patient is in the knee-bank position.

The Bony Pelvis.—The vertebral column articulates with the pelvis below through the *acroiliac articulation*. The *pubis* and *ischium* ligaments connect the pelvis with

contains fluid and gaseous intestinal content. The variation in sound both as to the frequency and intensity is great.

The most important use of abdominal auscultation is in the differentiation of *organic and paralytic ileus* (p. 1857). In the former, the peristaltic sounds may be greatly increased. In paralytic ileus the gurglings are feeble and infrequent. They may be absent over long periods of time.

During pregnancy the *uterine souffle* and the *fetal heart sounds* are audible. Occasionally a *rushing murmur* may be heard in the epigastrium in cases of ascites. This is a *venous hum* and is a sign of portal obstruction. *Continuous murmurs* with systolic accentuation are heard over vascular tumors and aneurysms of the abdominal aorta.

A patch of hair over the lower spine suggests the presence of *spina bifida occulta* (p 2800) or other congenital anomalies (p 2817) An unusually deep dimple at the lumbosacral junction may denote a *spondylolisthesis* (p 2970) A midline sinus in the sacrococcygeal region with a skin lined orifice and a protruding tuft of hair occurs with a *pilonidal sinus*

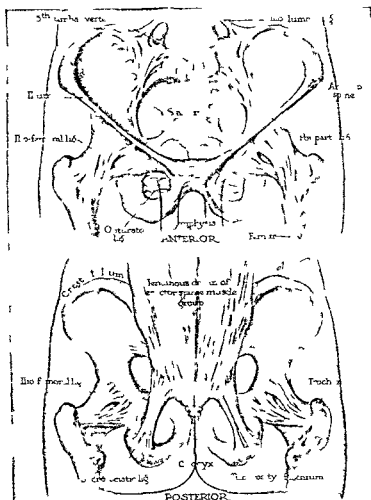


Fig 10 6—Bones and ligaments of the pelvis in the male*

(p 3045) *Spastic muscles* are often unduly prominent They may obliterate the normal lumbar lordosis

A total shift of the trunk to one side or the other (*sciatic scoliosis*) accompanies the sciatic syndrome of *low back pain* *Lumbar lordosis* should prompt additional examination to determine whether the curvature is structural or due to *psoas spasm* With the patient supine *psoas spasm* is suggested by an inability to extend the thigh completely on the trunk and

the vertebral column. The articulation itself is strengthened by the *anterior and posterior sacro iliac ligaments*.

The *sacro iliac joints* are true joints possessing a joint cavity. They allow very slight motion. The *symphysis pubis* is situated anteriorly and may allow slight motion especially during pregnancy.

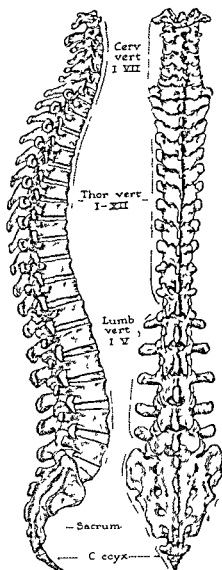


Fig 1035—The vertebral column as seen in lateral and posterior view

See *Differential Diagnosis of Commoner Febrile Skeletal Disorders* (p 193) *Disturbances of the Cervical Spine* (p 2818) *Swellings of Back* (p 2899) *Thoracic and Upper Lumbar Pain* (p 2940) *Fractures of Face Jaws Spine Pelvis and Thorax* (p 3003) *Scoliosis* (p 3060) *Kyphosis* (p 3069) *Low Back Pain* (p 3079)

CLINICAL EXAMINATION OF THE SPINE

Inspection—The examiner should note posture and muscular development curvatures and deformities elevation of a scapula or of one side of the pelvis and atrophy of muscle groups

• Jones and Shepard *Manual of Surgical Anatomy*

A patch of hair over the lower spine suggests the presence of *spina bifida occulta* (p 2820) or other congenital anomalies (p 2817) An unusually deep dimple at the lumbosacral junction may denote a *spondylolisthesis* (p 2970) A midline sinus in the sacrococcygeal region with a skin lined orifice and a protruding tuft of hair occurs with a *pilonidal sinus*

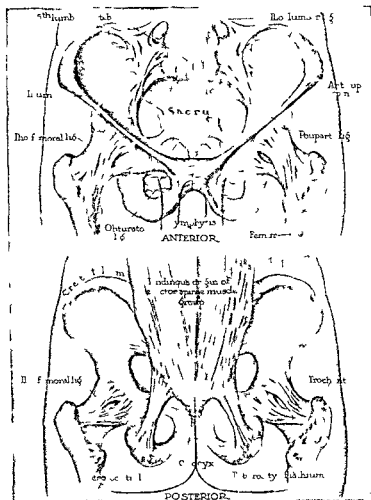


Fig 1036—Bones and ligaments of the pelvis in the male

(p 3945) *Spastic muscles* are often unduly prominent. They may obliterate the normal lumbar lordosis.

A total shift of the trunk to one side or the other (*sciatic scoliosis*) accompanies the sciatic syndrome of *low back pain*. *Lumbar lordosis* should prompt additional examination to determine whether the curvature is structural or due to *psoas spasm*. With the patient supine *psoas spasm* is suggested by an inability to extend the thigh completely on the trunk and

the tendency of the patient to lie with the corresponding hip flexed. Psoas spasm occurs with retroperitoneal inflammatory processes.

Lasègue and Patrick Signs—In the presence of disturbances in the lower part of the back and legs the Lasègue and Patrick tests are performed. The *Lasègue maneuver* consists in eliciting pain and resistance when the extended leg is flexed at the thigh. With disturbances of the sciatic nerve this produces pain due to stretching. The *Patrick sign* is elicited with the patient lying on his back. The affected limb is flexed at the hip and knee and the heel is placed on the opposite patella. The flexed knee is then pressed forcibly downward by the examiner whereupon pain occurs in the presence of any disturbance in the hip and pelvic joints.

Kernig and Brudzinski Signs—With evidences of intracranial or meningeal irritation the Kernig and Brudzinski signs are sought. The *Kernig sign* is the production of pain and resistance when the leg which is flexed at the hip is extended at the knee. The *Brudzinski sign* consists of flexion of the knees when the examiner passively flexes the head on the chest.

See *Differential Diagnosis of Pain and Stiffness of Neck* (p. 3020).

Movements of the Spine—The movements of the spinal column are forward flexion, backward extension, lateral deviation, and rotation. Anomalies of the spine often lead to postural deformities. Postural abnormalities produce anatomical alterations of the spine.

After inspection the spine should be put through its range of motion. The patient bends over and touches the floor. The normal spine flexes smoothly and completely into a rounded curve more prominent in the dorsal area. The lumbar lordosis should be completely obliterated and reversed. Oftentimes a segment a few vertebrae in length will break the smooth curve. This is best seen from the lateral view. *Rotational deformities* of the spine such as usually accompany structural scoliosis become apparent in this position as one side of the rib cage seems higher or more prominent than the other.

In *low back pain* (p. 3072) forward flexion is difficult and painful. The patient may note sharp pain in the first part of the curve or on arising from the bent over position. Motion should be tested in *hyperextension*, *lateral bending*, and *rotation*.

Palpation of the Spine—Palpation of the back for *tender spots* is done with the patient prone. As a rule *localized tenderness* indicates a lesion in the underlying region since the deep seated structures rarely give rise to visible surface changes.

The examination of the back should include a survey of the muscles of the back and the abdomen. The *abdominal muscle tone* should be noted in the standing position. Weakness is best demonstrated when the patient arises from the supine position.

The *corset* is examined in female patients. Heavy flabby women require boned supports with lacing. Visceroptotic patients may need a *hypo-gastric pad*.

CLINICAL EXAMINATION OF THE BONY PELVIS

The regions of the sacro iliac joints are examined for *indurations*, *painful nodules*, and *tenderness*. *Muscle spasm* and *list*ing accom any low

back strain (p 3063) The *symphysis pubis* is tested for mobility in the recently parous particularly if the obstetrician is known to practice symphysiotomy

THE UPPER EXTREMITIES

See *Differential Diagnosis of Disturbances of Ossification* (p 2798) *Increased Radiotranslucency of Bone* (p 2806) *Polyarthralgia* (p 2802) *Monarthralgia* (p 2803) *Diminished Motility of Joints Locking and*

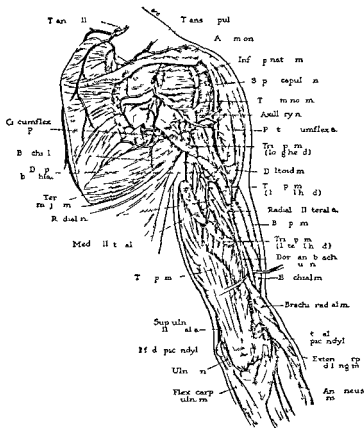


Fig 1037—Dissection of the deep structures of shoulder and arm posterior view. Portion of the deltoid, supraspinatus, infraspinatus, teres minor and triceps brachii muscles have been removed.

Ankylosis (p 2810) *Hypermotility and Abnormal Motility of Joints* (p 2808) *Ostealgia* (p 2841) *Swelling of Bone* (p 2844) *Pathologic Fractures* (p 2846) *Endocrinal Osteoarthropathies* (p 2846) *Metabolic Osteoarthropathies* (p 2878) *Disturbances of Muscle* (p 2882) *Pain in Shoulder and Upper Extremities* (p 2898) *Pain in Hands and Fingers* (p 2908) *Disturbances of Epiphyses* (p 2930) *Febrile Skeletal Disorders* (p 192) *Swellings of Shoulders and Upper Extremities* (p 2954) *Dis*

locations and Subluxations (p 2964), *Fractures of Shoulder and Upper Extremities* (p 3014)

TABLE 171 —PHYSICAL EXAMINATION OF THE UPPER EXTREMITIES

| Joint | Type | Movements |
|-------------------|-----------------|---|
| Shoulder | Ball and socket | Flexion backward extension lateral abduction adduction internal and external rotation circumduction |
| Axilla | | |
| Elbow | Hinge | Flexion and extension |
| Wrist | Hinge | Dorsiflexion palmar flexion radial deviation |
| Hands and Fingers | Hinge | Flexion extension apposition of thumb slight abduction and adduction in other digits |

THE LOWER EXTREMITIES

TABLE 172 —PHYSICAL EXAMINATION OF THE LOWER EXTREMITIES

| Joint | Type | Movements |
|----------------------------|--|---|
| Hip (Fig 1036) | Ball and socket | Flexion extension adduction abduction and external rotation |
| Groin | | |
| Knee | Hinge with menisci between articular surfaces patella in quadriceps tendon | Flexion and extension no lateral mobility |
| Legs | | |
| Ankle | Hinge | Dorsiflexion and plantar flexion inversion and eversion at subtalar joint |
| Mid tarsal Joints and Feet | Hinge | Twisting |
| Toes | Hinge | Flexion and extension |

See *Differential Diagnosis of Disturbances of Ossification* (p 2798)
Increased Radiotranslucency of Bone (p 2806) *Polyarthralgia* (p 2803)

Monarthralgia (p 2803) *Febrile Skeletal Disorders* (p 192) *Diminished Motility Locking and Ankylosis of Joints* (p 2810) *Hypermotility and Abnormal Motility of Joints* (p 2808) *Painless Swellings and Deformities*

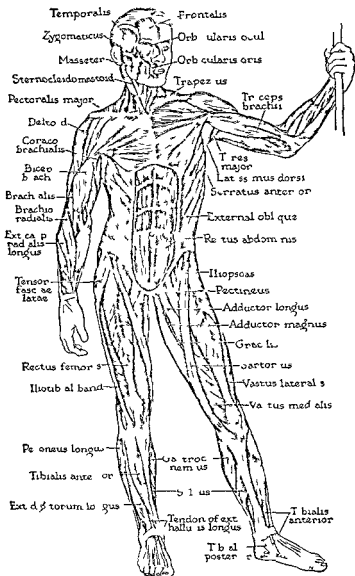


Fig 1036—Muscles of the body Anterior view

of Hips etc (p 2826) *Lump* (p 2832) *Ostealgia* (p 2841) *Swellings of Bone* (p 2844) *Pathologic Fractures* (p 2846) *Swellings of Inguinal Region* (p 3092) *Pain and Tenderness of Hips etc* (p 2869) *Endocrinal*

Osteo arthropathies (p 2856) *Metabolic Osteo arthropathies* (p 2863) *Disturbances of Muscle* (p 2882), *Pain and Tenderness of Hands Feet etc* (p 2908) *Disturbances of Epiphyses* (p 2930) *Dislocations and Subluxations* (p 2964), *Fractures of Hips and Lower Extremities* (p 3038)

THE PERIPHERAL VASCULATURE

The peripheral vasculature is best studied by direct observation of the retinal vessels using the *ophthalmoscope* (p 3628) The vessels of the rest of the body and the extremities may be examined by noninstrumental methods

ANATOMIC REVIEW

The Arteries—The arteries are cylindrical tubular vessels which serve to convey blood from the ventricles of the heart The distribution of the systemic arteries is like that of a highly ramified tree each branch being smaller than the trunk from which it arises In every instance the combined cross section area of the branches exceeds the cross section area of the parent vessel so that the surface area covered by the arterial tree is relatively enormous In their distribution arteries communicate with one another forming anastomoses Through this mechanism an effective collateral circulation may be established

Each artery consists of an *intima* or inner coat of lining endothelial cells a *middle muscular coat* also containing elastic and fibrous tissue and an *external fibrous coat* with the nutrient vessels nerves and lymphatics The arteries themselves are nourished by the *vasa vasorum* In addition there are distinct lymphatic vessels and lymphatic spaces in the vessel walls Myelinated and nonmyelinated nerve fibers surround the muscular coat which is richly supplied with vasomotor nerves The finest ramifications of the arteries and arterioles pass into the *capillaries* These are small endothelial tubes which connect the arterial and venous systems

The arterial system is divided into pulmonary and systemic groups

The Pulmonary Arteries—The pulmonary arteries convey *venous blood* from the heart to the lungs The main artery about 2 inches in length arises from the right ventricle in front of the aorta It divides into a right and left branch each of which supplies its respective lung The left pulmonary artery is connected to the under surface of the arch of the aorta by the *ligamentum arteriosum* This is the remnant of the *ductus arteriosus* of fetal life See *Patent Ductus Arteriosus* (p 207)

The Systemic Arteries—The main systemic artery is the *aorta* which originates from the upper part of the left ventricle and consists of an ascending arch and descending portion

The *ascending aorta* is about 2 inches in length It arises on a level with the lower border of the third left costal cartilage It passes obliquely upward forward and to the right The pulmonary artery is to the left of the ascending aorta the superior vena cava and the right auricle are to the right The only branches of the ascending aorta are the coronary arteries which supply the heart

The *arch or transverse aorta* situated at the level of the second costal cartilage passes upward backward and to the left in front of the trachea and finally passes downward on the left side of the body of the fourth thoracic vertebra where it becomes the descending portion The left vagus phrenic and superficial cardiac nerves pass in front of the arch The trachea the deep cardiac plexus the left recurrent nerve the esophagus and the thoracic duct lie behind Below the arch is the bifurcation of the pulmonary artery the superficial cardiac plexus the left recurrent nerve and the left bronchus

Three branches are given off from the arch of the aorta—the left common carotid the left subclavian and the innominate arteries The *innominate artery* divides into the right subclavian and right common carotid branches The *subclavian arteries* serve as the arterial stems for the upper extremities the *common carotids* divide into internal and external branches and supply the structures of the head and neck

The *descending aorta* is divided artificially into a thoracic and abdominal portion The *thoracic aorta* is contained in the back part of the posterior mediastinum It extends from the lower border of the fourth thoracic vertebra on the left to the aortic opening in the diaphragm in front of the lower border of the last thoracic vertebra It gives off visceral branches to the bronchi the esophagus the pericardium and the mediastinal structures There are also

parietal branch lies to the muscles and rigid structures of the thoracic cage. The *abdominal aorta* commences at the aortic opening of the diaphragm. At the level of the fourth lumbar vertebra it divides into the two *common iliac arteries*. Through the *celiac axis* it gives off branches to the stomach, liver and spleen through the *superior mesenteric artery* to the small intestines, the right and left adrenals, the right and left kidneys and the right and left gonads. The large bowel is supplied through the *inferior mesenteric artery*. The parietal struc-

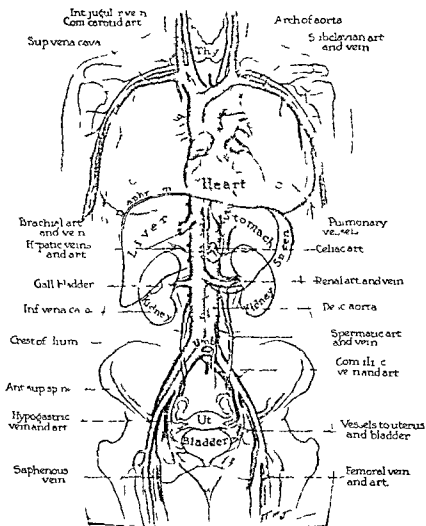


Fig. 1039.—Heart and principal blood vessels in the female

ture are nourished by individual branches from the first, second, third and fourth lumbar regions.

The *common iliac arteries* are about 2 inches in length and each divides into an internal and external iliac artery. The *internal iliac* supplies the bladder and rectum as well as the uterus and vagina in the female. It then divides into an internal pudic artery which supplies the perineum and male genitalia and a sacrotuberous and sacral artery which supplies the back of the

thigh The external iliac artery passes beneath Poupart's ligament to enter the thigh as the femoral artery which furnishes the blood supply of the lower extremity

The walls of the arteries are innervated by the involuntary nervous system Stimulation of adrenergic fibers produces powerful constriction and angospasm with elevation of systemic blood pressure

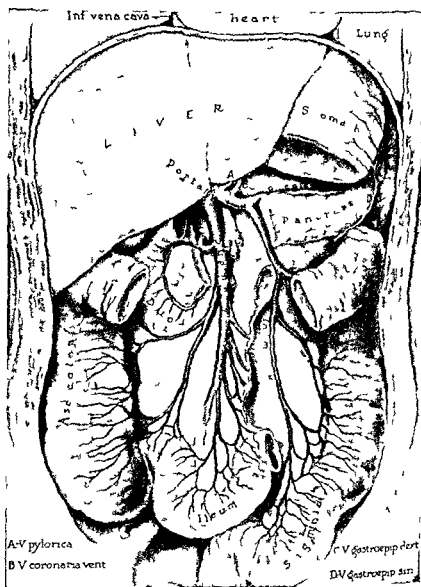


Fig 1010—The portal system *

The Veins—The veins convey the capillary blood from the periphery back to the heart. The veins begin as minute plexuses receiving blood from the capillaries. These unite into trunks, each trunk being of greater caliber than any of its branches. The cross section of the trunk, however, in every instance is less than the sum of the cross sections of its tributaries. As a general rule, the veins run together with the arteries. The accompanying veins are always larger and more numerous than the arteries. Their walls, while histologically the same, are thinner due to smaller amounts of elastic and muscular tissue. As in the case of the

arteries the veins communicate very freely with each other forming large collateral circulations. Unlike the arteries the veins are equipped with valves which prevent backflow.

The Pulmonary Veins—The pulmonary veins return arterial blood from the lungs to the left auricle. They are four in number, two for each lung. They contain no valves.

Systemic Veins—The systemic veins are divided into four groups: (1) the cardiac; (2) those of the head and neck, upper extremities and thorax which terminate in the superior vena cava; (3) those of the lower extremities, abdomen and pelvis which terminate in the inferior vena cava; and (4) the portal system.

1 The *cardiac veins* are essentially the left and right coronaries which follow the course of the arteries. The two main vessels join to form the coronary sinus, about 1 inch in length, which terminates in the right auricle.

2 The veins of the *head and neck* which follow the arteries join to form the *internal jugular veins*, each of which in its turn unites with the subclavian veins after they have received the external jugulars to form the *right and left innominate veins*. The latter crosses the arch of the aorta to form the *superior vena cava* by union with the right innominate.

3 The blood from the lower extremities is collected through the femoral, external and internal iliac veins and the common iliacs which unite to form the *inferior vena cava*. Both the great caval vessels then enter the right auricle to discharge the return venous blood into the heart.

4 Accessory to the inferior caval system is the *portal system*. The portal vein is formed by the junction of the splenic, superior mesenteric, gastric pyloric, cystic and para-umbilical veins. The portal system drains the spleen, stomach, pancreas, the colon, rectum and pelvis, the small intestine and the gallbladder. It is 3 inches in length and enters the liver through a right and left branch. Here the blood is again distributed through a system of smaller radicles, once more to be collected by the hepatic veins by which the blood is conveyed to the inferior vena cava. The blood from the portal system thus passes through two sets of ramifications.

Lymphatics—The lymphatic system comprises a network of fine vessels which collect fluid from the tissue spaces. These are present in the skin and subcutaneous tissues and possibly though not definitely in the muscles as well. These delicate tubules unite to form larger but still slender ducts which again divide into smaller channels as they approach lymph nodes. Beyond each node the channels again become united and proceed to the next node station. Eventually the lymphatic flow empties into the venous circulation via the subclavian veins or the thoracic duct.

See: *The Circulatory System* (p. 771)

EXAMINATION OF PERIPHERAL VESSELS AND LYMPHATICS

By simple inspection and palpation of the peripheral vessels and lymphatics a great wealth of information is available. Thickening and increased pulsation of the peripheral arteries may be demonstrable in arterio-sclerotic disturbances (p. 976). Engorgement and pulsation of the jugular vein accompany backward failure (p. 941). The carotid artery often throbs inordinately with systolic hypertension (p. 910) and increased pulse pressure (p. 918). Palpation of the radial pulse is most often used for counting cardiac rate and for determining the regularity of cardiac rhythm. With advanced peripheral arterial disease thickening and beading of the wall may seem apparent when the vessel is compressed against the lower end of the radius. Radial pulsations should not be used for estimation of arterial tension since readings with the sphygmomanometer are much more accurate.

In the lower extremity the femoral artery is readily palpated. Occasionally it is the site of an aneurysm. The popliteal artery is felt with difficulty unless the knee is flexed. Examination of the legs and feet is not complete unless an attempt is made to palpate both anterior and posterior tibial vessels. Peripheral vascular disease is usually more marked in the lower than in the upper extremities (p. 994). Demonstration of reason

DIFFERENTIAL DIAGNOSIS OF

Abnormalities of the Pulse

Considerable information may be obtained by inspection or palpation of temporal, radial, posterior tibial and dorsalis pedis arteries. Oscillometry (p 791) is of some assistance in estimating changes in the leg vessels but the sphygmograph is no longer worth the trouble involved in its technical execution.

PHYSICAL SIGNS CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|-----------------------------------|--|
| Tortuous Temporals | Generalized arteriosclerosis (p 981) Temporal arteritis with predominant headache (p 1512) |
| Reduced in Both Arms | Aortic aneurysm (p 798) Look for localized pulsation by fluoroscopy. Note dilated arch on radiograph. Get Wassermann reaction. |
| Reduced in One Arm | Scalenus anticus syndrome with pain in the arm relieved by relaxation of the muscle (p 2953). Consider surgery. |
| Reduced in Both Legs | Coarctation of aorta with marked differences in pressure readings between arms and legs (p 959). Peripheral arteriosclerosis with demonstrable calcification on radiographs (p 981). Monckeberg's sclerosis with beading of vessels (p 994). Thrombo-angitis obliterans with history of recurrent phlebitis and intractable pain (p 1029). |
| Tenderness of Peripheral Arteries | Periarteritis nodosa with protracted low grade fever and eosinophilia. Get biopsy (p 1027). |
| Thickening of the Vessels | Generalized arteriosclerosis (p 981). Thrombo-angitis obliterans with history of recurrent phlebitis and intractable pain (p 1029). Periarteritis nodosa with protracted low-grade fever and eosinophilia (p 1027). Monckeberg's sclerosis with beading of the vessels (p 994). |
| Bounding Pulse | With fever or nervousness. Following exercise. In neurocirculatory asthenia with absence of objective findings (p 897). Hyperthyroidism with elevation of basal metabolic rate and therapeutic response to iodide (p 1197). Aortic insufficiency with diastolic murmur and high pulse pressure (p 970). Hypertension with elevations of both systolic and diastolic pressures (p 900). After injections of epinephrine (p 3876). |
| Water Hammer Pulse | Corrigan phenomenon in hyperthyroidism, anemia, aortic insufficiency and with arterial venous fistulas. Seek diastolic murmur at base. Get basal metabolic rate and hemogram. |
| Plateau Pulse | Aortic stenosis with rough systolic murmur at base and low pulse pressure (p 971). Pericardial effusion and cardiac tamponade with gradual widening of area of precordial flatness and obliteration of apex impulse. Consider diagnostic pericardiocentesis (p 1197). |

| | |
|-------------------------|---|
| Dicrotism | Typhoid fever with protracted pyrexia lymphocytosis bacteremia or positive Widal reactions (p 225) |
| Bisferiens | Double impulse with each heart beat In aortic stenosis with roughened systolic murmur at base and low pulse pressure (p 971) In marked hypertension with elevation of systolic and diastolic pressures (p 900) In advanced generalized arteriosclerosis (p 981) |
| Pulsus Paradoxicus | Softening or obliteration of pulse during inspiration. In cardiac tamponade pericardial effusion adhesive pericarditis and with mediastinal tumor Note increase in precordial flatness Get chest x ray Consider diagnostic pericardiocentesis (p 852) |
| Pulsus Alternans | Alternating strong and weak beats Myelomalacia with marked Ecg changes (p 992) Frequent premature contractions (p 887) |
| Capillary Pulse | Hyperthyroidism with elevation of basal metabolic rate (p 1197) Aortic insufficiency with diastolic murmur at base (p 970) |
| Reduction of All Pulses | Ergotism as seen in patients sensitive to ergot amine taken for the relief of migraine (p 1506) |

ably full and equal pulses in the tibial vessels suggests but does not absolutely prove the integrity of the arterial supply to lower limbs inability to demonstrate satisfactory pulsation is not necessarily a definite indication of arterial disease Many elderly patients reveal normal circulatory phenomena in feet and toes despite inability to demonstrate pulsations in the tibial vessels Discrepancy between the vessels of the two sides points to significant unilateral arterial disease

The examination of the peripheral vessel includes a survey of the visible mucous membranes of the conjunctival sac and the lids With pallor or brick redness a hemogram is required to reveal the possible presence of an anemia or a polycythemia (p 1092) ashen cyanosis accompanies forward failure in shock and syncope (p 998) a deep purple blueness characterizes backward failure and disturbances of aeration in the pulmonary alveoli due to disease of the respiratory system or to poisoning in which the hemoglobin molecule is deprived of an adequate quota of oxygen (p 3827)

The examination of the peripheral vascular system is completed by a search for varicosities in the scrotum (varicocele) and legs particularly over the shin areas The presence of edema warrants intensive investigation (p 706) since it connotes profound disturbances of water balance

METHODS OF EXAMINATION FOR ARTERIAL ADEQUACY

Color Changes—Arterial insufficiency is indicated by color changes that follow alteration in the position of an extremity The patient rests exposed in a warm room for ten to fifteen minutes The hands and arms

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METHODS OF EXAMINATION FOR ARTERIAL ADEQUACY

Color Changes—Arterial insufficiency is indicated by color changes that follow alteration in the position of an extremity. The patient rests exposed in a warm room for ten to fifteen minutes. The hands and arms

are then raised above the head and the fingers are rapidly clenched and released. With normal arterial circulation there is slight blanching following this maneuver. If the arterial circulation is embarrassed the skin becomes extremely pale and at times waxy. One finger may show greater pallor than the others. When the hands resume their normal dependent position the skin of the normal person returns to its previous color usu-

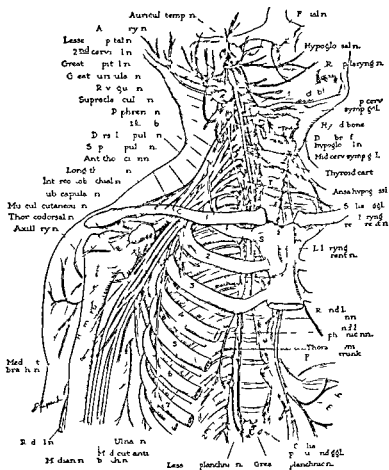


Fig 1041.—The right cervical and brachial plexuses of the systemic (yellow) and sympathetic (blue) nerves and their branches shown in relation to skeleton and body outline

ally within five seconds. Delayed reappearance of normal color suggests impaired arterial function.

To test the lower extremities the patient lies supine and raises the legs to a position at right angles with the trunk. The toes are flexed and the ankles are moved in extension and flexion for one half minute. Here again as in the arms the patient with arterial impairment will show excessive and at times irregular pallor. The feet are lowered and the patient sits on the edge of the bed or table with legs dependent. The color normally re-

turns in 10 seconds but may take as long as 45 seconds if the arteries are inadequate

Venous Filling Time—An indirect method of estimating arterial adequacy is by testing the venous filling time. The extremity to be examined is elevated until the veins collapse. It is then quickly lowered to the dependent position. If the veins fail to fill within ten seconds, arterial insufficiency is likely.

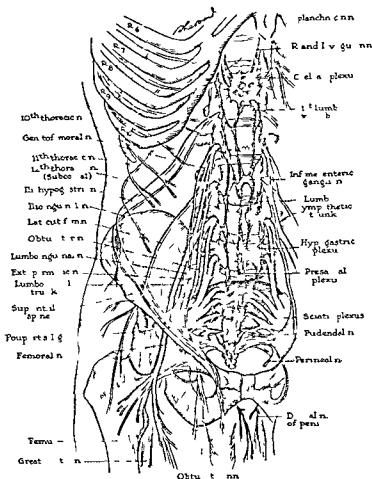


Fig 1042—The right lumbar and sacral plexuses of system c (yellow) and sympathetic (blue) nerves and their branches shown in relation to the skeleton

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THE NERVOUS SYSTEM

During the course of the examination the practitioner may obtain considerable information regarding the functions of the nervous system. The presence of any abnormality merits specialist consultation.

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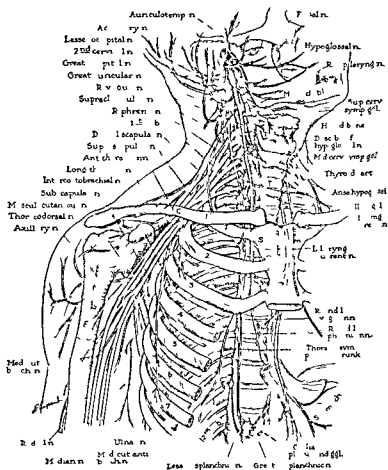


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 DIFFERENTIAL DIAGNOSIS OF

Ataxia

The differential diagnosis of disturbances of gait, other than ataxia, is elsewhere considered (p 2736). The present table deals only with the causes for unsteadiness.

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|--|--|
| Pharmacodynamic | Alcoholism (p 3848). Overdosage with sedatives and hypnosis (p 3842). |
| Hypoglycemia | Spontaneous due to pancreatic adenomas (p 1262). Overdosage with insulin (p 1237). Late effect of administration of glucose. |
| Ménière's Syndrome | With nystagmus and vomiting. Sudden onset and relief (p 1485). |
| Postural Hypotension | On sudden change of position from recumbent to standing (p 916). Note pressure changes. |
| Otogenic | With labyrinthitis and cerebellar ataxias. Usual history of preceding otitis (p 2146). Look for chronic aural discharge. Refer to otologist. |
| Ophthalmic | In conditions associated with nystagmus. Refer to ophthalmologist. |
| Muscular | Following fatigue or atrophy as from prolonged bed rest or immobilization in plaster. |
| Hysteria | Ataxia-abasia with normal physical status (p 1308). Refer to psychiatrist. |
| Peripheral Neuritis | Plot areas of anesthesia to touch, pain, vibratory sense, heat and cold. Get cerebrospinal fluid. Refer to neurologist (p 148b). |
| Tuber Dorsalis | with pupillary irregularity, absent deep reflexes and postural findings in cerebrospinal fluid (p 1464). Treat intensively with penicillin. |
| Multiple (Disseminated) Sclerosis | Remittent neuropathy between twentieth and thirty-fifth years. Frequently manifested by diplopia, pallor of temporal half of optic disc and hyperreflexia (p 1504). Get spinal fluid and refer to neurologist. |
| Subacute Combined Degeneration of Cord | With patchy areas of involvement. Get spinal fluid. Refer to neurologist. |
| Friedreich's Ataxia | Hereditary disturbance with athetosis and speech disturbance (p 1415). Refer to neurologist. |
| Thrombosis of Posterior Inferior Cerebellar Artery | Hemiataxia with hetero-lateral diminution of sensations of pain, heat and cold (p 1444). Refer to neurologist. |
| Hemiplegia | With contra-lateral spastic paralysis and hyperreflexia (p 1439). Refer to neurologist (p 1471). |
| Cerebellar Tumors | Unilateral or bilateral ataxia. Not increased by closing eyes. Reflexes normal. Gait reeling (p 1426). Refer to neurologist. |

TABLE 173—ROUTINE SURVEY OF THE NERVOUS SYSTEM

| Structure or Function | Method of Examination |
|--|--|
| Speech | Note during history |
| Gait | Note on entering room |
| Muscle Strength and Coordination | Note while undressing weakness paralysis asymmetry atrophy hypertrophy tremor athetosis tics |
| Abdominal Reflexes | Compare both sides |
| Cremasteric Reflex | Compare both sides |
| Triceps Jerk | Strike tendon with elbow at a right angle forearm extends compare both sides |
| Biceps Jerk | Strike tendon with elbow at a right angle forearm flexes compare both sides |
| Patellar Reflex (Knee Jerk) | Strike tendon with knee at a right angle leg extends reinforce by instructing patient to clasp hands when tendon is struck (Jendrassik) compare both sides |
| Achilles Reflex (Ankle Jerk) | Strike tendon while foot is held by examiner in dorsiflexion foot plantar flexes compare both sides |
| Plantar Reflex (Babinski) | Stroke outer side of sole of foot, upward and inward normal response is plantar flexion of all toes abnormal response is dorsiflexion of great toe and plantar flexion with fanning of remaining toes compare both sides |
| Plantar Reflex (Oppenheim) | Stroke medial side of tibia downward toward ankle responses as with Babinski |
| Plantar Reflex (Gordon) | Compress the calf muscles responses as with Babinski |
| Plantar Reflex (Chaddock) | Stroke external malleolus responses as with Babinski |
| Clonus | Briskly dorsiflex foot while leg is held firm repetition of ankle jerk |
| Touch | Test with cotton |
| Pain | Test with pin |
| Tactile Discrimination | Test with head and point of pin |
| Vibratory Sense | Test with tuning fork placed over tibia olecranon clavicle or sternum |
| Olfactory Nerve | Smell peppermint or camphor |
| Optic Nerve | Ophthalmoscopy visual acuity visual fields |
| Oculomotor Trochlear and Abducens Nerves | Associated eye movements ptosis of lid strabismus nystagmus pupillary responses to light and accommodation |
| Trigeminus | Sensations of face corneal reflex power of jaw muscles |
| Facial Nerve | Test facial muscles wrinkle forehead taste of salt and sugar on anterior portion of tongue |
| Auditory Nerve | Test hearing with watch and tuning fork note nystagmus |
| Glossopharyngeus | Gag reflex sensation of soft palate taste on back of tongue |
| Vagus | Cardiac rate voice production swallowing |
| Spinal Accessory | Shrug shoulders |
| Hypoglossal | Protrude tongue |

| | |
|-------------------------------|--|
| Metallic Speech | Paralysis of recurrent laryngeal nerve Often due to aortic aneurysm (p 798) Get laryngoscopy and chest x ray |
| Feminization in the Male | Hypogonadism (p 2412) |
| Masculinization in the Female | Following androgen therapy (p 2404) In Cushing's syndrome (p 1159) arrhenoblastoma of ovary malignant neoplasms of adrenal cortex and other masculinizing tumors (p 2481) At menopause (p 2493) |
| Mutism | Congenital abnormality associated with deafness (p 1310) |
| Central Aphasia | Destruction of speech center in dominant cerebral lobe due to tumor abscess hemorrhage thrombosis or embolism Hysteria (p 1353) Get complete neurologic status and refer patient to neurologist. |

Integrations for Nervous System

See *Differential Diagnosis of Disturbances of the Gait* (p 2496) *Disturbances of Gait in Childhood* (p 2736) *Disturbances of Speech* (p 3556) *Disturbances of Muscle* (p 2682) *Disturbances of the Abdominal Wall* (p 3556) *Disturbances of Decubitus* (p 3494) *Disturbances of the Head in Adults* (p 2504) *Disturbances of the Fontanelles* (p 2729) *Disturbances of the Head in Infancy* (p 2774) *Convulsions in Infancy* (p 1519) *Convulsion in Adults* (p 1519) *Headache* (p 1512) *States of Coma and Unconsciousness* (p 1294) *Feeble-mindedness* (p 1333) *Disturbances of the Cerebrum* (p 1428) *Disturbance of the Spinal Cord* (p 1432) *Abnormalities of the Face* (p 3506) *Pain in and Stiffness of the Neck* (p 320) *Table 98 Reaction pattern of Injury to the Peripheral Nerve Cord and Tract* (p 1494) *Table 99 Peripheral Nerve Injuries* (p 1458) *Table 99 Disturbances of the Pterice* (p 1494) *Fig 230 Cutaneous Field of the Peripheral Nerve (Posterior View)* (p 1494) *Fig 231 Cutaneous Field of the Peripheral Nerve (Lateral View)* (p 1495) *Fig 232 Cutaneous Fields of the Peripheral Nerve (Anterior View)* (p 1496) *Fig 233 Cutaneous Fields of the Peripheral Nerve (Perineal View)* (p 1497) *Olfactory Neuritis* (p 1490) *Optic Neuritis* (p 1646) *Oculomotor Trochlear and Abducens Neuritis* (p 1645) *Trigeminal Neuritis* (p 1490) *Facial Neuralgia* (p 1480) *Auditory Neuritis (Deafness)* (p 2019) *Glossopharyngeal Neuritis* (p 1488) *Vagus Neuritis* (p 1395) *Neuritis of the Spinal Accessory Nerve* (p 1490) *Hypoglossal Neuritis* (p 1489) *Table 91 Participation of the Involuntary Nervous System in Clinical Disturbances* (p 1306)

 DIFFERENTIAL DIAGNOSIS OF

Disorders of Speech

Disorders of speech require differentiation from disturbances of voice production due to abnormalities of the larynx (p 3496) Speech disorders may be of trivial importance as in tongue-tie they may indicate grave lesions as in central aphasia due to destruction of the speech center The main problem of the practitioner is to differentiate lesser difficulties from the more profound lesions The latter require consultation with the neurologist for further exploration and therapy

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|-----------------------------------|--|
| Lisping | Tongue-tie Note restriction due to short frenum |
| Improper Pronunciation of Labials | Hairlip and cleft palate (p 1586) Refer for plastic surgery |
| Nasal Twang | Edentia Nasal obstruction due to rhinitis adenoids or persistent discharge Tonsillar hypertrophy Glossitis Dislocation of jaw Peritonsillar abscess Epidemic parotitis Stomatitis Cleft palate Postdiphtheritic bulbar palsy Make careful inspection of oropharynx |
| Stammering and Stuttering | Usually psychogenic Consider reference to psychiatrist |
| Delayed or Defective Speech | In infancy With mental deficiency idiocy and imbecility Consider consultation with neurologist In adults The more severe types of dementia general paresis and cerebral arteriosclerosis Get cerebrospinal fluid and consider reference to neurologist |
| dioglossia | Baby talk Usually imitative |
| Echolalia | Babbling talk in severe types of mental deficiency (p 1332) Refer to psychiatrist |
| Palilalia | Pathological repetition of words In mental deficiency and the severer types of dementia Refer to psychiatrist |
| Scanning Speech | Multiple sclerosis with patchy areas of sensory disturbance pallor of temporal half of optic disc and hyperreflexia (p 1504) Hereditary cerebellar ataxia |
| Explosive Speech | Friedreich's ataxia (p 1415) Spastic paralysis (p 1455) |
| Slurring Speech | General paresis with positive spinal fluid (p 1377) Alcoholism (p 3848) |
| Jerky Speech | Chorea (p 190) Get temperature record and sedimentation rate Look for evidences of carditis |
| Slow Hesitating Speech | Myxedema with low basal metabolic rate and therapeutic response to thyroid extract (p 1193) Mental deficiency (p 1337) Huntington's chorea (p 1417) |
| Monotonous Speech | Paralysis agitans with intention tremor festination and mask like facies (p 1505) |
| Running Down Speech | Myasthenia gravis with therapeutic response to neostigmine (p 2886) |

| | |
|-------------------------------|---|
| Metallic Speech | Paralysis of recurrent laryngeal nerve Often due to aortic aneurysm (p 798) Get laryngoscopy and chest x ray |
| Feminization in the Male | Hypogonadism (p 2412) |
| Masculinization in the Female | Following androgen therapy (p 2404) In Cushing's syndrome (p 1150) arrhenoblastoma of ovary malignant neoplasms of adrenal cortex and other masculinizing tumors (p 2481) At menopause (p 2493) |
| Mutism | Congenital abnormality associated with deafness (p 1310) |
| Central Aphasia | Destruction of speech center in dominant cerebral lobe due to tumor abscess hemorrhage thrombosis or embolism Hysteria (p 1353) Get complete neurologic status and refer patient to neurologist |

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DIFFERENTIAL DIAGNOSIS OF

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The Nasal Mucosa—The nasal mucosa or *schnneiderian membrane* is covered with respiratory epithelium. In the olfactory region which comprises the uppermost portion of the nasal septum and the medial surfaces of the superior and inferior turbinates is found the specialized olfactory epithelium which has to do with reception of the sense of smell. The *schnneiderian membrane* extends anteriorly to the skin of the nasal vestibule and posteriorly to the respiratory mucosa of the nasopharynx. It is continued into the various nasal sinuses.

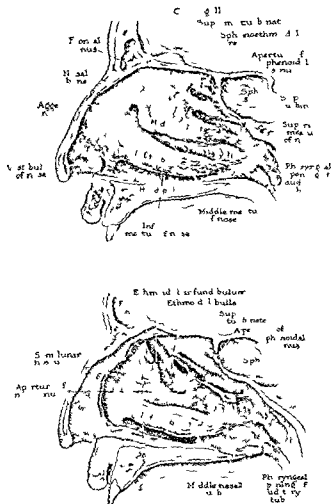


Fig 1043—Upper figure lateral wall of right nasal cavity. Lower figure same with middle turbinate removed. Maxillary sinus shown by dotted lines.

through their offices through the lacrimal apparatus to the conjunctiva and through the eustachian tube to the tympanum and mastoid cells. These ramifications of the membrane are important in the understanding of the extensions of nasal infection by continuity and contiguity (p 2148).

The Nerves—The nose is innervated by the first and fifth cranial nerves. The olfactory or first cranial nerve is sensitive to smell. Ordinary sensation is transmitted from the nose for the most part through the trigeminal nerve which possesses a ganglionic enlargement.

CHAPTER 162

THE PHYSICAL EXAMINATION DARK ROOM

The body cavities are best examined with electrodiagnostic equipment in a dark room. In the office a closet suffices for the investigation; in the home of the patient a blanket may be used as a cover to exclude light.

ELECTRODIAGNOSTIC EQUIPMENT

Examinations may be made by direct illumination using a pocket torch or by indirect illumination provided by the head mirror. Electrodiagnostic sets add immeasurably to the range and accuracy of investigations; the cost of the equipment is relatively nominal considering its wide usefulness and durability. Upkeep is negligible as the bulbs last for many months and repairs are rarely required.

Electrodiagnostic sets may operate on flashlight batteries or house current. The latter requires the purchase of a rheostat which assures adaptability of the strength of illumination. The equipment is portable and may be used in the home of the patient as well as in the office.

ELECTRODIAGNOSTIC EXAMINATIONS

| | |
|---|--|
| Inspection of Nose (p. 3592) | Myringoscopy (p. 3610) |
| Anterior Rhinoscopy (p. 3592) | Inspection of Eye and Adnexa (p. 3622) |
| Transillumination of Nasal Accessory Sinuses (p. 3595) | Ocular Motility (p. 3622) |
| Inspection of Lips, Mouth, Buccal Mucosa, Palate, Oral Cavity, Gums, Teeth and Tongue (p. 3595) | Pupillary Reactions (p. 3623) |
| Inspection of Oropharynx and Tonsillar Ring (p. 3599) | Ophthalmoscopy (p. 3623) |
| Indirect Laryngoscopy (p. 3603) | Transillumination of Eyeball, Breast, Scrotum and Subcutaneous Tissues (p. 3632) |
| Inspection of Auditory Canal and External Ear (p. 3609) | Proctoscopy (p. 1907) |
| | Sigmoidoscopy (p. 1907) |
| | Vaginoscopy (p. 3647) |

For the interpretation of the significance of findings it is necessary to review the anatomy of investigated structures. The information obtained by the practitioner often requires amplification by the specialist consultant.

THE NOSE

ANATOMIC REVIEW

The nose is the special organ of smell. Its pyramidal shape is maintained by a cartilage and bone foundation covered externally by skin. Internally it is lined by mucous membrane and is divided into two chambers by a bony and cartilaginous *septum*. The most anterior portion of the nose is the *vestibule*. The external openings are the *nares* while the posterior openings into the nasopharynx are the *choanae*.

The Turbinates and the Meatuses.—The lateral nasal walls present three irregular prominences: the *superior middle* and *inferior turbinates*. Together with the roof and the floor of the nose the turbinates form the *superior middle* and *inferior meatuses*. The *accessory sinuses* drain into the meatuses: the posterior ethmoid and sphenoid into the superior and the frontal maxillary and anterior ethmoid sinuses into the middle meatus. The inferior meatus contains the orifice of the nasolacrimal duct.

The Nasal Mucosa—The nasal mucosa or *schnneiderian membrane* is covered with respiratory epithelium. In the olfactory region which comprises the uppermost portion of the nasal septum and the medial surfaces of the superior and inferior turbinates is found the specialized olfactory epithelium which has to do with reception of the sense of smell. The *schnneiderian membrane* extends anteriorly to the skin of the nasal vestibule and posteriorly to the respiratory mucosa of the nasopharynx. It is continued into the various nasal sinuses.

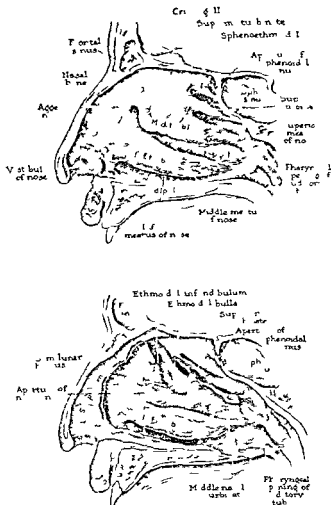


Fig 1043—Upper figure lateral wall of right nasal cavity. Lower figure same with middle turbinate removed. Maxillary sinus shown by dotted lines.

through the canaliculi through the lacrimal apparatus to the conjunctiva and through the eustachian tube to the tympanum and mastoid cells. These ramifications of the membrane are important in the understanding of the extensions of nasal infection by continuity and contiguity (p. 2148).

The Nerve—The nose is innervated by the first and fifth cranial nerves. The olfactory or first cranial nerve is sensitive to smell. Ordinary sensation is transmitted from the nose for the most part through the *trigeminal nerve* which possesses a ganglion enlargement.

DIFFERENTIAL DIAGNOSIS OF

Internal Abnormalities of the Nose

Internal abnormalities of the nose are revealed by direct inspection and anterior rhinoscopy (p 3592) Almost invariably they are associated with snoring nasal obstruction mouth breathing nasal discharge and epistaxis (p 2193) Successful relief of symptoms may be accomplished by intranasal manipulations or surgery

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|------------------------------|---|
| Cysts | At nasal vestibule or naso-labial junction (p 2066) Aspiration reveals clear fluid |
| Foreign Bodies | Usually unilateral (p 2046) Accompanied by putrid or hemorrhagic discharge Demonstrate by anterior rhinoscopy Refer to specialist for removal |
| Fractures | May be compounded with tear of mucous membrane and subcutaneous emphysema (p 3013) Avoid blowing of nose Get x ray |
| Furunculosis | Usually situated at roots of vibrissae Also associated with intense pain and swelling (p 2109) Institute vigorous antibiotic therapy because of imminent danger of intracranial complications or bacteremia |
| Gumma | Painless elastic swelling Usually situated at vestibule or septum (p 2046) Get Wassermann Note therapeutic response to iodide |
| Malignant Neoplasms | Rare cancerous or sarcomatous growths with progression in size ulceration and bleeding (p 2067) Get biopsy |
| Abscess of Nasal Septum | Usually painful and associated with obstruction (p 2122) Start vigorous antibiotic therapy Prepare for incision. |
| Deviation of Nasal Septum | May be asymptomatic or accompanied by occlusion and hypertrophy of impinging turbinate Often associated with secondary accessory nasal sinusitis (p 2126) Get x ray Consider reparative surgery |
| Hematoma of Nasal Septum | Following trauma May be associated with fracture Get x ray |
| Perforation of Nasal Septum | Usually traumatic or operative but may be syphilitic Get Wassermann (p 2046) |
| Ulcer of Nasal Septum | May be associated with recurrent epistaxis (p 2193) Lesions may be local or a manifestation of tuberculosis leprosy glanders or syphilis Get Wassermann. Attempt identification of pathogen by local spread or culture (p 52) |
| Papilloma | May be solitary or multiple Often asymptomatic but may produce nasal obstruction and facial deformity Frequent epistaxis (p 2066) Refer for intranasal removal |
| Atresia of Posterior Choanae | Congenital anomaly causing nasal obstruction. Demonstrate by attempt to pass catheter (p 2043) Refer to specialist for intranasal surgery |
| Acute Rhinitis | The common cold (p 2114) Early congestion with rhinorrhea Later purulent discharge occurs |

| | |
|--------------------------------|--|
| Atrophic Rhinitis | Glazing of nasal mucosa with crusting and foul odor (p 2122) Refer to specialist for investigation of sinuses |
| Hypertrophic Rhinitis | Chronic inflammatory thickening of nasal mucous membrane Often associated with polyps (p 2121) Refer to specialist for investigation of nasal accessory sinuses (p 2125) |
| Membranous Rhinitis | May be acute or chronic Associated with irritating nasal discharge Get smears and culture for <i>C. diphtheriae</i> (p 302) Prepare for intensive serum therapy |
| Non seasonal Allergic Rhinitis | Usually a bacterial allergy Associated with sneezing nasal itching and rhinorrhea (p 2096) Investigate nasal accessory sinuses Get cultures Prepare for skin testing and desensitization (p 563) |
| Seasonal Rhinitis | Hay fever with intense congestion edema and rhinorrhea (p 2097) Discharge may contain eosinophils Prepare for skin testing and desensitization (p 2097) |

(the *sphenopalatine ganglion*) situated deep in the sphenomaxillary fossa Irritation of the ganglion may produce severe pain and discomfort, relieved by local application

Blood Vessels and Lymphatics—Vasomotor nerves controlled by a vasomotor center in the medulla supply the vessels of the mucosa and its erectile tissue These are probably connected with nuclei of the vagus through association fibers

The *blood supply* of the external nose consists of branches of the internal and external maxillary and the ophthalmic arteries The veins terminate in the anterior facial and ophthalmic vessels The nasal septum receives its arterial supply from the anterior and posterior ethmoidal arteries the ophthalmic and posterior septal branches of the sphenopalatine The veins empty into the ophthalmic vein and the pterygoid plexus The lateral wall of the nose is supplied by the anterior and posterior ethmoidal arteries and lateral nasal arteries from the sphenopalatine

Some of the *nasal venous channels* pass through the cribriform plate of the ethmoid bone to communicate with veins in the interior of the skull Thus local infections of the Schneiderian membrane may produce widespread and distant disturbances through contiguity of mucous surfaces and by way of the venous channels

Large venous plexuses exist in the submucosa of the inferior turbinate and constitute the erectile tissue of the nose These respond delicately to emotional and physical stimuli through fibers of the involuntary nervous system

The *lymphatics* of the external nose drain into cranial facial and submaxillary nodes From the nasal cavity the deep cervical chain of lymphatics connects with the deep pharyngeal nodes and the deep cervical nodes Lymphatics of the nasal sinuses drain into the retropharyngeal nodes These channels provide routes for infection in *retropharyngeal abscess* and *cervical lymphadenopathy*

Functions of the Nose—The *olfactory function* of the nose is performed by branches of the first nerve distributed in the superior meatus

The nasal chambers and their accessory cavities are of prime importance in *voice production* Nasal obstruction unfavorably affects the quality of the voice Most singers and orators have well developed nasal resonance While the tones are initiated by vibrations of the vocal cords overtones are produced in the resonating chambers of the nose and accessory nasal sinuses

The *respiratory function* of the nose is most important since it prepares the air for the normal exchange of oxygen and carbon dioxide in the walls of the air vesicles The air is warmed to body temperature preventing irritation of the mucosa and the air vesicles of the lower respiratory tract It is filtered by the cilia and moistened by the mucosa of the turbinate bones These properties are important factors in removing foreign and infectious

matter from the respiratory passages Destruction of these protective mechanisms by well intended application of bactericides (p 2027) is most unfortunate

Examination of the Nose—Examinations of the nose are limited to investigation of its patency and to intranasal inspection

Patency of Airway—The patency of the airway in each nostril is tested by closing the opposite nostril with the finger and directing the patient to shut the mouth and forcibly exhale through the free naris

Anterior Rhinoscopy—The anterior portion of the nasal septum is examined for *ulcers* and *perforations* A small septal ulcer usually results from nasal picking and is a common cause of epistaxis Infections of the nasal vibrissae may cause painful and extensive swelling The mucosa may be atrophic hypertrophic or polypoid

Inspection of the nose by anterior and posterior rhinoscopy may reveal the presence of *secretion* in the vicinity of the natural orifices of the various paranasal sinuses After this secretion is removed *congestion* and *edema* of

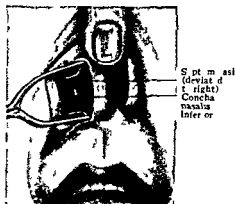


Fig 1044—Anterior rhinoscopic examination *

the turbinates or changes in the mucosa surrounding the orifices may be visualized The exploration is facilitated by spraying the mucosa with a mild anesthetic and vasoconstrictor solution (0.5 per cent pontocaine with a few drops of neosynephrine or 4 per cent cocaine with 3 per cent ephedrine)

After shrinkage of the mucosa the interior of the nasal cavity may be more carefully examined The reappearance of gross secretion should be noted If the secretion is in the middle meatus the involved sinus may be the frontal the anterior ethmoid or the antrum If the secretion appears above the middle turbinate between the septum and the superior turbinate it comes from the sphenoid or posterior ethmoidal cells For the determination of the exact origin of the secretion the patient is referred to the specialist who performs *posterior rhinoscopy* *diagnostic sinus lavage* and interprets *radiographs* (p 2023)

See *Differential Diagnosis of Pain in the Nose* (p 2067) *Disturbances of Smell* (p 2120) *Sneezing* (p 2064) *Epistaxis* (p 2123)

* Callender Surgical Anatomy

THE NASAL SINUSES

Presumptive evidence of infection of the nasal sinuses is obtained by the appearance of intranasal secretion or post nasal discharge and by *transillumination*. Specialist procedures include *diagnostic lavage* and *radiography*.

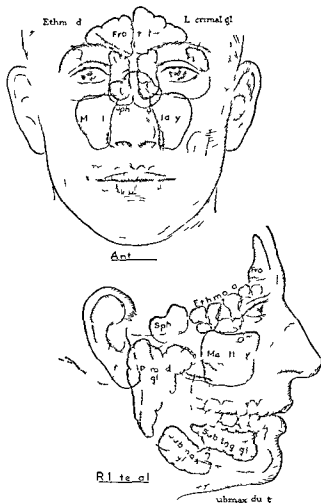


Fig 1045—Accessory sinuses of the nose projected on face in anterior and lateral views. Sphenoidal and L. maxillary sinuses also shown.

ANATOMIC REVIEW

The nasal accessory sinuses are bilateral and usually consist of four on each side: the frontal, ethmoidal, maxillary and sphenoidal. Each is lined with nasal mucous membrane, contains air, and communicates with the nasal fossa through its natural orifice. The anterior group of sinuses drains into the middle meatus; the posterior group into the superior meatus.

The Frontal Sinuses—The frontal sinuses lie behind the superciliary arches and are

usually asymmetrical. They are usually not present at birth making their first appearance about the fourth year and developing to full size after puberty. After the eighth year they become of clinical importance because they have attained a definite size and cavity. Boas septa usually subdivide them into various compartments. The sinuses vary greatly in size and form in many instances one of them differs in extent and shape from the one on the opposite side. Occasionally one may be rudimentary or entirely absent.

The frontal sinuses communicate with the middle meatus by means of the frontonasal passage. Of importance is their proximity to the meninges and the brain above and the orbit below. Infectious processes occasionally spread in one or the other direction.

The Ethmoid Sinuses—The *ethmoid sinuses* each of which is divided into anterior, middle and posterior cells lie between the upper portion of the nose and the orbit. Only a thin lamina of bone separates them from orbit and meninges so that infection may readily travel by contiguity. They open into the middle and superior meatuses. Ethmoidal cells are present at birth. They do not thoroughly develop until puberty. They are of variable size and number and are situated between the medial wall of the orbit and the superior and middle turbinate bones. Occasionally ethmoidal (turbinal) cells are found within the middle turbinate fossa.

The *bullae ethmoidalis* is a prominence caused by an ethmoidal cell of the anterior group. Frequently a large ethmoidal cell may extend into the frontal sinus. The attachment of the middle turbinate marks the line of division between the anterior and posterior groups of cells, the anterior lying in front and below and the posterior above and behind. The posterior cells are fewer in number and larger in size than the anterior. There may be between seven and eleven ethmoidal cells usually arranged in single file from anterior to posterior. The posterior cells may encroach upon the sphenoidal sinus. They may extend over the orbital bone as orbital extensions into the sphenoid. They occasionally extend behind the antrum.

The Maxillary Sinuses (Antrums of Highmore)—The *maxillary sinuses* while present at birth do not attain full size until after the eruption of the permanent teeth. The maximum development is attained between the fifteenth and eighteenth years. The maxillary sinuses are the largest of nasal accessory sinuses and are irregular shaped pyramids with their bases presenting to the nasal fossa and their apices lying in the zygomatic process of the maxillae. The medial wall of each sinus or base is formed by the ethmoid bone, the maxillary process of the inferior turbinate and a small portion of the lacrimal bone. The roof separates the cavity from the orbit. The floor is formed by the alveolar portion of the superior maxilla in the outer part of the hard palate. The maxillary antrum communicates with the middle meatus by means of the *ostium maxillare*. An accessory ostium is often present posterior to the natural opening.

The second bicuspid and the first or second molars are in close relation to the floor of the maxillary sinuses. These sometimes project into the bony cavities at which point the bone may be thin. A suppurative process around a tooth may affect the mucous membranes of the sinus. Upon extraction of the tooth an alveolar fistula communicating with the maxillary sinus is produced. The roof of the maxillary sinus contains the infraorbital nerve. This is covered by mucous membrane and may be injured during the course of a radical maxillary sinus operation thus causing persistent neuralgia (p. 1489).

The Sphenoid Sinuses—The sphenoid sinuses are usually absent before the fourth year and not fully developed until about the tenth. They are bilateral and may be of unequal size depending on pneumatization of one sphenoid bone at the expense of the other. They are separated by the intrasphenoid septum. The posterior walls and roofs of the sphenoids may be cancellous or thin.

The sphenoid sinuses communicate with the superior meatus by a small aperture which empties into the spheno-ethmoid region. The openings of the sphenoid sinuses are invariably membranous since their bony apertures are much larger than their actual membranous openings. The superior walls of the sphenoid sinuses are in relation to the cranial cavity especially the sella turcica containing the pituitary bodies and the optic nerves. The sphenoid sinus when pneumatized is separated from the cavernous sinus by a thin bone. Delicacies of the lateral wall of the sphenoid establish continuity of the sphenoid mucosa with the dura forming the wall of the cavernous sinus and with the carotid artery the ophthalmic division of the fifth nerve and the third, fourth and sixth nerves. The optic nerve may be in close proximity to the sphenoid sinus where it is vulnerable to neuritis in cases of sphenoidal sinus infection. The vidian nerve (a branch of the maxillary) crosses through the body of the sphenoid bone in the region of the floor of the sphenoid sinus and may become irritated because of infection.

The relation of the sphenoid bone to the meninges cranial nerves and brain should be noted since extensive sphenoid suppuration may result in *rhinogenic meningitis* (p 2128) *optic neuritis* *brain abscess* *sphenopalatine* or *vidian neuralgia* and *carotid sinus thrombosis*

Transillumination of the Sinuses.—The frontal and maxillary sinuses are examined by transillumination with the electrodiagnostic set

For transillumination of the *frontal sinuses* the beam of light is placed in the upper inner angle of the orbit The passage of the ray is compared on the two sides *Differences in illumination* are of some clinical significance particularly if they are accompanied by unilateral pain and tenderness or discharge However differences may be due to inequality in the sizes of the sinuses and in the relative thickness of the frontal bones

The *maxillary antrum* is transilluminated from within by placing the beam of light in the patient's mouth after which the lips are closed Normally the maxillae light up and a *retinal reflex* is obtained Transillumination of the maxillary antrum from without is accomplished by instructing the patient to open the mouth and tilt back the head The illumination of the hard palate is observed when the beam of light is placed over the cheek externally

Differential alterations are of more importance than diminished radiability on both sides In *acute sinusitis* before the membrane has thickened or there is considerable discharge transillumination may be perfectly clear while active inflammation is going on Contrariwise when there has been *chronic thickening* of the wall there may be poor transillumination of the sinus and yet no active or important clinical sinus disease may be present

The same limitations exist for radiography as transillumination so that when there is question concerning the sinuses the patient should be referred to the rhinologist for a diagnostic lavage (p 2023)

See *Differential Diagnosis of Pain in the Face* (p 2130) *Sneezing* (p 2064) *Pain in the Nose* (p 2067) *Nasal Discharge* (p 2100) *Cough* (p 2050) *Disturbances of Smell* (p 2120) *Epistaxis* (p 2123) *Headache* (p 1512) *Cryptogenic Fevers* (pp 26 2760) *Rhinogenic Brain Abscess* (p 2129) *Rhinogenic Meningitis* (p 2128) *Sphenopalatine Neuralgia* (p 2148) *Vidian Neuralgia* (p 1483) *Retrobulbar Neuritis* (p 1640) *Orbital Cellulitis* (p 2130)

THE LIPS

The lips oral cavity and pharynx are accessible to direct inspection Disturbances of the gums and teeth are the province of the dentist while pharyngeal lesions are referred if necessary to the rhinolaryngologist

Because of the thinness of the easily visible mucous membrane of the lip circulatory changes such as *pallor* *capillary pulsation* and *cyanosis* may be observed earliest in this situation

See *Differential Diagnosis of Breath Odors* (p 1660) *Disturbances of the Lip* (p 1685)

THE MOUTH

The mouth is divided into a smaller anterior vestibule and the oral cavity proper

The Buccal Mucosa—The buccal mucosa forms the inner lining of the cheek At a point opposite the second molar tooth of the upper jaw is a

usually asymmetrical. They are usually not present at birth making their first appearance about the fourth year and developing to full size after puberty. After the eighth year they become of clinical importance because they have attained a definite size and cavity. Bony septa usually subdivide them into various compartments. The sinuses vary greatly in size and form in many instances one of them differs in extent and shape from the one on the opposite side. Occasionally one may be rudimentary or entirely absent.

The frontal sinuses communicate with the middle meatus by means of the frontonasal passage. Of importance is their proximity to the meninges and the brain above and the orbit below. Infectious processes occasionally spread in one or the other direction.

The Ethmoid Sinuses—The *ethmoid sinuses* each of which is divided into anterior, middle and posterior cells lie between the upper portion of the nose and the orbit. Only a thin lamina of bone separates them from orbit and meninges so that infection may readily travel by contiguity. They open into the middle and superior meatuses. Ethmoidal cells are present at birth. They do not thoroughly develop until puberty. They are of variable size and number and are situated between the medial wall of the orbit and the superior and middle turbinate bones. Occasionally ethmoidal (turbinal) cells are found within the middle turbinate fossa.

The *bulia ethmoidalis* is a prominence caused by an ethmoidal cell of the anterior group. Frequently a large ethmoidal cell may extend into the frontal sinus. The attachment of the middle turbinate marks the line of division between the anterior and posterior groups of cells. The anterior lying in front and below and the posterior above and behind. The posterior cells are fewer in number and larger in size than the anterior. There may be between seven and eleven ethmoidal cells usually arranged in single file from anterior to posterior. The posterior cells may encroach upon the sphenoidal sinus. They may extend over the orbital bone as orbital extensions into the sphenoid. They occasionally extend behind the antrum.

The Maxillary Sinuses (Antrums of Highmore)—The *maxillary sinuses* while present at birth do not attain full size until after the eruption of the permanent teeth. The maximum development is attained between the fifteenth and eighteenth years. The maxillary sinuses are the largest of nasal accessory sinuses and are irregular shaped pyramids with their bases presenting to the nasal fossa and their apices lying in the zygomatic process of the maxillae. The medial wall of each sinus or base is formed by the ethmoid bone. The maxillary process of the inferior turbinate and a small portion of the lacrimal bone. The roof separates the cavity from the orbit. The floor is formed by the alveolar portion of the superior maxilla in the outer part of the hard palate. The maxillary antrum communicates with the middle meatus by means of the *ostium maxillare*. An accessory *ostium* is often present posterior to the natural opening.

The second bicuspid and the first or second molars are in close relation to the floor of the maxillary sinuses. These sometimes project into the bony cavities at which point the bone may be thin. A suppurative process around a tooth may affect the mucous membranes of the sinus. Upon extraction of the tooth an alveolar fistula communicating with the maxillary sinus is produced. The roof of the maxillary sinus contains the infraorbital nerve. This is covered by mucous membrane and may be injured during the course of a radical maxillary sinus operation thus causing persistent *neuralgia* (p. 1482).

The Sphenoid Sinuses—The sphenoid sinuses are usually absent before the fourth year and not fully developed until about the tenth. They are bilateral and may be of unequal size depending on pneumatization of one sphenoid bone at the expense of the other. They are separated by the intrasphenoid septum. The posterior walls and roofs of the sphenoids may be cancellous or thin.

The sphenoid sinuses communicate with the superior meatus by a small aperture which empties into the sphenoid-ethmoid region. The openings of the sphenoid sinuses are invariably membranous since their bony apertures are much larger than their actual membranous openings. The superior walls of the sphenoid sinuses are in relation to the cranial cavity especially the sella turcica containing the pituitary bodies and the optic nerves. The sphenoid sinus when pneumatized is separated from the cavernous sinus by a thin bone. Dehiscences of the lateral wall of the sphenoid establish continuity of the sphenoid mucosa with the dura forming the wall of the cavernous sinus and with the carotid artery, the ophthalmic division of the fifth nerve and the third, fourth and sixth nerves. The optic nerve may be in close proximity to the sphenoid sinus where it is vulnerable to neuritis in cases of sphenoidal sinus infection. The vidian nerve (a branch of the maxillary) crosses through the body of the sphenoid bone in the region of the floor of the sphenoid sinus and may become irritated because of infection.

Through premature loss of the first teeth or through faulty habits which produce bony changes by sustained or habitual pressure the normal pattern is often disturbed resulting in overcrowding and *malocclusion* (p 1703)

The *crown of the tooth* consists of a shell of inorganic *enamel* enveloping the less vitreous *dentin*. Centrally located and running vertically

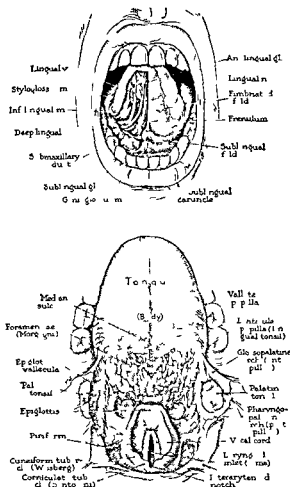


Fig 1046—Above Under surface of tongue with right side dissected to show principal muscles blood vessels and nerves (Eycleshymer and Jones) Below The tongue from above showing adjacent structures of the pharynx and larynx

toward the root is a channel which houses the *dental pulp* a complex mass of nerves blood vessels lymphatics and cellular elements

The *root* consists largely of dentin encased in a thin coat of bone-like *cementum*. The pulp continues its course through the center of the root to the apex beyond which it communicates with the vascular and nervous

papilla the summit of which presents the opening of the parotid duct (Stensen)

Eruptions may occur on the buccal mucosa. These may be purely local in their origin or they may represent enanthems of the acute *exanthematous diseases* (p 172)

The buccal mucosa may be the site of brownish black *pigmented areas*. While these develop in patients with *Addison's disease* (p 1271) they may be without clinical significance in dark skinned races

The Palate—The *hard palate* is a firm arched structure extending from the gums and teeth of the maxilla to the soft palate. The hard palate forms the anterior two thirds of the roof of the mouth. Radiating laterally from the midline are several prominent linear ridges the *rugae*. The hard palate may be the site of the congenital malformation of *cleft palate* (p 1686)

The *soft palate* and *uvula* are easily viewed when the mouth is open. The mucous membrane is pink and soft. The uvula is situated in the midline. It should not be sufficiently long to impinge upon the base of the tongue or the posterior pharyngeal wall. Local inflammatory processes in the nasopharynx are apt to give rise to an alarming *edema of the uvula*. The swelling may actually block the *urway* so that scarification is necessary

The Gums—The gums consist of dense mucosa strongly adherent to the periosteum. They cover the alveolar arches of the maxilla and mandible and surround the necks of the teeth. Even in health the margins of the gums are sufficiently separated from the tooth surfaces to form superficial crevices which habitually harbor detritus and microorganisms. The surfaces of the gums are studded with minute mucous glands. They are normally firm and pink

The Teeth—The teeth of the *first dentition* are smaller and whiter than the permanent teeth. These *deciduous* or *milk teeth* are 20 in number and consist of two lower and two upper central incisors, two upper lateral and two lower lateral incisors, four anterior molars, four canines (cuspids) and four posterior molars. The average time of eruption in the order above mentioned varies between the fifth and thirtieth month

There are 32 teeth in the *secondary* or *permanent dentition*. The four first molars generally appear in the sixth year, eight incisors in the sixth, seventh and eighth years, four bicuspid (premolars) in the ninth, four canines (cuspids) in the twelfth year, and eight second molars—four of which are bicuspid (second premolars)—by the thirteenth birthday. In addition four third molars complete the adult set sometime after the sixteenth year. Through the stimulus of their eruption on bone formation there is a forward growth of the maxilla and mandible. The third molars are frequently impacted because of insufficient room in the jaws

In certain individuals the third molars remain unerupted and give rise to considerable difficulty necessitating extraction. Delay and irregularities in the order of the appearance of the teeth usually result from malnutrition or from mental and metabolic defects

In the completed dentition the teeth of each jaw are arranged in the pattern of a semi oval. The cusps and fossas of the biting surfaces of the posterior teeth interlock when the jaws are closed. The upper anterior teeth extend slightly in front of and over the lower anterior teeth

Through premature loss of the first teeth or through faulty habits which produce bony changes by sustained or habitual pressure the normal pattern is often disturbed resulting in overcrowding and *malocclusion* (p 1703)

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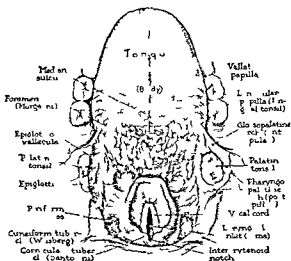
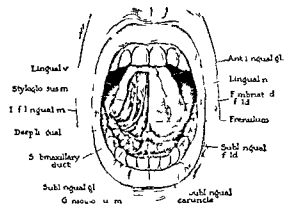


Fig 1016—Above Under surface of tongue with right side dissected to show principal muscles blood vessels and nerves (Eycleshymer and Jones) Below The tongue from above showing adjacent structures of the pharynx and larynx

toward the root is a channel which houses the *dental pulp* a complex mass of nerves blood vessels lymphatics and cellular elements

The *root* consists largely of dentin encased in a thin coat of bone-like *cementum*. The pulp continues its course through the center of the root to the apex beyond which it communicates with the vascular and nervous

systems Each root is embedded in a *socket* of the *alveolar process* of the mandible or maxilla Separating the root from bone is a *fibro elastic periodontal membrane* which permits a slight movement of the tooth during mastication and serves as a shock absorbing cushion to both structures

The Tongue—The tongue is concerned with the sense of *taste* It assists in *mastication* *deglutition* and *phonation* It is a muscular organ with a wide range of mobility

The body of the tongue is convex on its dorsal surface and divided centrally by a raphe The dorsum is studded with several forms of *papillae* The larger *circumvallate papillae* are few and arranged in a V shaped line posteriorly They contain a large number of taste buds The *fungiform papillae* occur on the lateral borders tip and dorsum These undergo early atrophy in *hyperchromic anemia* (p 1077) The *filiform papillae* are scattered over the dorsal tip Both serous and mucous glands are liberally distributed between the papillae

The ventral surface of the tongue is smooth and glistening The superficial branches of the ranine veins are clearly delineated beneath the mucosa The under surface of the tongue is attached to the lower gums and floor of the mouth At the midline is the *lingual frenum* a fibromuscular strand which limits the mobility of the organ Anterior to the frenum is a *bifid papilla* that contains the orifices of the submaxillary and sublingual salivary glands The mucous membrane in this region is thin and the vascularity is great *sublingual administration* (p 3807) of a drug is almost the equivalent of hypodermic injection

Innervation—The muscles of the tongue are innervated by the hypoglossal (twelfth) nerve With unilateral paralysis (p 1488) the involved muscles atrophy and as the tongue is protruded the tip deviates to the paralyzed side

The sensory nerves of the tongue are (1) the trigeminal (2) the chorda tympani of the facial (seventh) and (3) the glossopharyngeal (eleventh) The sensation of touch is conveyed by the trigeminal nerve Taste travels by the chorda tympani from the anterior two thirds of the tongue and by the glossopharyngeus from the posterior one third

The Salivary Glands—Three pairs of glands contribute to the salivary secretion The largest the *parotid* lies in the substance of the cheek overlying the angle of the mandible in front of the external ear Its excretory channel *Stensen's duct* pierces the *buccinator muscle* and opens within a small papilla on the inner buccal surface

Intermediate in size the *submaxillary gland* is situated centrally in the submaxillary triangle extending well under the border of the mandible *Wharton's duct* runs forward and upward with its proximal half lying quite superficially and terminates in a papilla lying anterior to the lingual frenum

The *sublingual gland* lies just within the arch of the body of the mandible and directly below the mucous surface adjacent to the median line It communicates with the floor of the mouth in this region via several ducts some of which merge with Wharton's duct

See *Differential Diagnosis of Breath Odors* (p 1660) *Enanthems of Oral and Buccal Surfaces* (p 1668) *Toothache* (p 1680) *Disturbances of the Tongue* (p 1687) *Gums* (p 1701) *Teeth* (p 1705) *Taw* (p

1705) *Palate* (1716) *Ptyalism* (p 1708) *Dysphagia and Pain on Swallowing* (p 1723)

THE PHARYNX

The pharynx is the expanded entrance of the alimentary tract which extends from the base of the skull above to the cricoid cartilage below. It is partially bisected by the soft palate to form the *nasopharynx* or *epipharynx* above and the true pharynx below. The latter is further subdivided into the *oropharynx* and the *laryngo* or *hypopharynx*. Examination of the nasopharynx requires an expert but the oropharynx is directly visible.

Functions of the Pharynx—The chief function of the nasopharynx is as a respiratory passage admitting the passage of air freely during inspiration and expiration. Because of its rigid walls the nasopharynx is shut off from the pharynx by the pulling up of the soft palate against the posterior pharyngeal walls during an act of swallowing and in certain phases of phonation. The nasopharynx helps in the ventilation of the eustachian tubes and tympanic cavities and forms a channel for drainage from the normal and pathologic nasal sinuses.

Secretions from the lacrimal ducts, the tympanic cavities and the eustachian tubes may also find their way into the nasopharynx.

The oropharynx and hypopharynx have to do with the act of swallowing and the processes of respiration and digestion. The mechanism of swallowing consists in the passage of the food from the mouth by the contraction of the tonsillar pillars and the movement of the tongue. The nasopharynx is shut off by the contraction and elevation of the soft palate while the larynx is closed by the rolling backwards of the base of the tongue to compress the epiglottis against the arytenoid. Meanwhile the glottis itself is closed by the intrinsic adductor muscles. The constrictor muscles of the pharynx pass the food into the esophagus the status of which opens momentarily as the food enters.

Innervation of the Pharynx—The pharynx normally is sensitive to pressure. Many patients have an increased gag reflex so that the local examination is exceedingly difficult. *Absence of the gag reflex* is supposedly an indication of hysteria.

The pharynx and uvula receive motor innervation through the *glossopharyngeal nerve*. Paralysis of the nerve such as occurs in diphtheria results in the nasal regurgitation of food and the characteristic nasal voice. With *bilateral paralysis* the soft palate and uvula are limp and flaccid. With *unilateral paralysis* the uvula points away from the side of the lesion.

The Nasopharynx—The nasopharynx is a dome shaped cavity the roof of which is formed by the inferior surface of the body of the sphenoid bone and the floor by the soft palate. The anterior wall contains the posterior choanae by which the nasal cavity communicates with the nasopharynx. The lateral walls are occupied by the pharyngeal openings of the eustachian tubes and the fossa of Rosenmüller which extends above and lateral to the eustachian orifice.

The vault of the nasopharynx is difficult to visualize. *Posterior rhinoscopy* requires an expert. Digital exploration of the nasopharynx is mentioned only to be condemned. It is dangerous to the examiner who may have a finger bitten and it is an ordeal for the patient yielding little satisfactory result or information.

The vault of the nasopharynx commonly contains variable amounts of *adenoid tissue*. When enlarged and infected these may block the choanae and cause post nasal obstruction and discharge with *chronic cough*.

The Oropharynx—The oropharynx lies behind the oral cavity from which it is separated by the tonsillar pillars and fossae above and the base of the tongue below. Above the soft palate separates it from the epipharynx. On the base of the tongue is the foramen caecum from which

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|------------------------------|---|
| Syphilis | Chancere of lip tongue pharynx or tonsils Confirm by darkfield microscopy (p 45) Secondary syphilid of pharynx or tonsils Confirm by darkfield microscopy and serology (p 45) Start intensive antibiotic treatment (p 340) |
| Fusosp rochetosis | Ulcerogangrenous infection with Vincent organisms demonstrable by direct smear Start intensive antibiotic therapy (p 355) |
| Other Respiratory Infections | Diffuse redness with normal oropharyngeal flora demonstrable bacteriologically (p 52) |
| Influenza | Diffuse redness with generalized myalgias (p 396) |
| Measles | Koplik spot of buccal mucosa followed by diffuse redness Look for morbilliform erup- tion Attempt abortive treatment with globulin (p 409) |
| Rubella | German measles with diffuse redness mor- billiform or scarlatiniform eruption posterior cervical lymphadenopathy and minor febrile reaction (p 417) |
| Varicella | Diffuse redness and vesiculation Note pleo- morphic monolocular eruption (p 420) |
| Varola | Generalized redness and vesiculation Note monomorphic polylocular eruption (p 424) |
| Infectious Mononucleosis | Diffuse inflammation with characteristic hemo- gram (p 466) Note atypical mononuclear cells positive heterophile reaction and false positive Wassermann test (p 336) |
| Tonsillitis | Diffuse redness of throat with generalized pains and evidences of meningeal irritation (p 457) Get cerebrospinal fluid |
| Virus Encephalides | Diffuse redness with evidences of meningeal irritation Get cerebrospinal fluid (p 449) |
| Mycoses | Chronic follicular or granulomatous lesions with demonstrable monilia actinomyces or coccidioides (p 485) |
| Neurogenic | Paralysis of uvula following diphtheria or facial and bulbar palsy Get neurologic status (p 3584) |
| Analogues of the Dermatoses | Particularly pemphigus with bullous lesions (p 3334) |
| Allergy | Angioneurotic edema of uvula or pharynx (p 3350) Prepare for scarification if airway is compromised Summon specialist for tracheot- omy if indicated |
| Postnasal Drainage | Seek evidences of adenoid hypertrophy chronic rhinitis or accessory nasal sinu- itis Get radiographs Refer to specialist for naso- pharyngoscopy and lavage of sinuses (p 2023) |
| Blood Dyscrasias | Hemorrhagic and ulcerogangrenous lesions in the leukemias and agranulocytic angina (p 1096) Get hemogram (p 3704) Consider examination of sternal marrow if spread is not diagnostic (p 1035) |

 DIFFERENTIAL DIAGNOSIS OF

Visible Abnormalities of the Throat

Visible abnormalities of the throat include painless as well as the painful conditions described in a previous chart (p 2071). Diagnosis is aided by the ease with which structures may be inspected and the facility with which specimens can be obtained for laboratory examination.

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|---|--|
| Congenital Malformations | Elongated, bifid or bulbous uvula (p 2043) Consider removal if indicated. |
| Mechanical Disturbances | Foreign bodies of pharynx or tonsils particularly fishbones. Calculus of tonsils (p 215 ^m) Consider tonsillectomy |
| Neoplasm | Juvenile fibroma of nasopharynx (p 2066) Malignant lympho-epithelioma of nasopharynx. Refer to specialist for nasopharyngoscopy and biopsy (p 2068). Carcinoma of uvula or epiglottis (p 2070). Lymphosarcoma or carcinoma of tonsil (p 2070). Refer to specialist for biopsy |
| Lymphoid Hyperplasia | Adenoids demonstrated by nasopharyngoscopy (p 2140). Hypertrophy of lingual tonsil, faucial tonsil and of remainder of pharyngeal lymphoid ring (p 2157). Consider lingual and faucial tonsillectomy and adenoidectomy if there is obstruction to airway or repeated infection |
| Local Inflammations | Acute and chronic nasopharyngitis, adenoiditis, pharyngitis, lingual tonsillitis, faucial tonsillitis and epiglottitis (p 2139). Obtain smears and cultures if infection is more than moderately severe or protracted. Consider antibiotic therapy |
| Abscess Formation | Thornwaldt abscess of nasopharynx (p 2140). Tonsillar, peritonsillar or retropharyngeal abscess (p 2155). Give intensive antibiotic therapy (p 106). Guard against obstruction to airway (p 2014). Be prepared for emergency treatment with tracheotomy if indicated (p 3951). Summon specialist for incision and drainage |
| Local Manifestations of Systemic Infection | |
| Streptococcal | Septic sore throat. Often with follicular exudate. Give intensive antibiotic treatment (p 106). Look for scarlatiniform eruption (p 180). Examine for evidences of rheumatic carditis (p 186) |
| Scarlet Fever | Diffuse redness followed by follicular pharyngotonsillitis. Note eruption verified by blanching reaction (p 177). Combine antibiotic treatment with antitoxin (p 106) |
| Diphtheria | Membranous exudate with Co. diphtheriae demonstrable by smear or culture. Combine antibiotic therapy with antitoxin (p 302) |
| Meningococcal Meningitis | Diffuse redness of throat with hemorrhagic eruption and evidences of meningeal irritation (p 206). Get blood culture and cerebrospinal fluid. Give intensive treatment with antibiotics (p 106) |

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site the thyroid gland originally made its descent and where occasionally *aberrant thyroid tissue* is found. The mucous membrane is normally smooth and pink and may have scattered islands of lymphoid tissue particularly in those who were subjected to a tonsillectomy early in life. These lymphoid

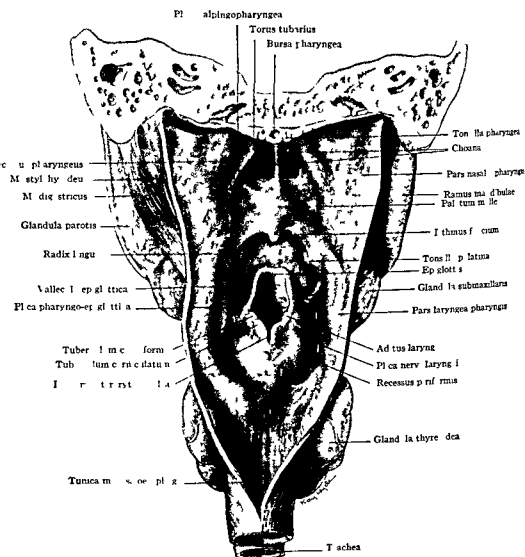


Fig 1047.—Posterior view of the pharynx and esophagus. The posterior wall of the pharynx is opened.*

phoid follicles may become infected and pass through the stages ordinarily observed in *follicular tonsillitis*.

The *lingual tonsils* are situated behind the base of the tongue and are surrounded by the circumvallate papillae. The lingual tonsil easily seen with a mirror may be hypertrophied to the size of the thumb and it may overhang the epiglottis. Under these circumstances particularly when it is infected it is a common cause of *intractable cough*.

Laterally in the oropharynx are found the palatine tonsils lying between the tonsillar pillars. The posterior wall is formed by the cervical vertebrae and on either side are vertical bands of lymphatic tissue the lateral pharyngeal masses which extend into the epipharynx. Occasionally on the posterior wall small lymphatic nodules may be found.

The Hypopharynx—The hypopharynx lies behind the larynx. Above it communicates with the oropharynx but narrows below to open into the esophagus by a transverse slit closed by the inferior constrictor muscle or cricopharyngeus. The anterior wall consists of the epiglottis the aryepiglottic folds the pyriform recess and the posterior surfaces of the arytenoid and cricoid cartilages. The posterior wall is formed by the cervical vertebrae and laterally is the pharyngeal musculature.

The Lymphatic Structure of the Pharynx—The pharyngeal lymphatic ring of Waldeyer consists of the lateral laryngeal folds the pharyngeal islands the lingual tonsils the palatine tonsils the pharyngeal tonsil or adenoid tissue and the lymphoid tissue around the eustachian orifices and in the fossa of Rosenmüller.

The *pharyngeal tonsil* or *adenoid* is present at birth and may atrophy after puberty if infection is not present. The adenoid is formed by three or four masses of lymphoid tissue separated by vertical clefts. There may be additional lymphoid tissue attached to the eustachian tube orifices and the fossa of Rosenmüller.

The adenoid mass or pharyngeal tonsil is situated on the roof and posterior wall of the epipharynx and occupies a variable area dependent upon its size. It is composed of lymphatic tissue and has a definite external morphology consisting of a median raphe and two or three lateral indentations which divide the adenoid mass into distinct folds. Exactly in the midline at the junction of the basisphenoid and the occipital bone is a small pit called the pharyngeal bursa. This is the site of the so called Thornwaldt's abscess (p. 2140).

The *palatine* or *faucial* tonsils are masses of lymphoid tissue embedded in a fossa formed by the anterior and posterior tonsillar pillars and the tongue. The *plica triangularis* is a fold of mucosa running from the anterior pillar to the tongue. It may cover the lower pole of the tonsil from which it may need to be separated in doing a complete tonsillectomy. The pharyngeal surface of the tonsils contains crypts which may extend to the capsule of the tonsil at its point of attachment. This attachment is formed by a fibrous capsule which separates the tonsil from the superior constrictor muscle. The blood supply of the tonsils comes from the palatine and tonsillar branches of the external maxillary artery the ascending pharyngeal branch of the external carotid and the lingual branches of the lingual artery. Hemorrhage from these vessels presents a major problem. The veins enter the pharyngeal venous plexus. The nerve supply arises from the sphenopalatine ganglion by way of the posterior palatine division above and a branch of the glossopharyngeal nerve through the lower pole. Lymphatic vessels pierce the capsules to run into the deeper cervical chain behind and below the mandible. The faucial tonsils are observed while the tongue is retracted and the patient makes the sound ah. Buried infected tonsils and tonsillar stumps remaining after incomplete tonsillectomy may not be observed unless the anterior pillar is retracted. Under

no circumstances should the tonsil be squeezed. This useless and needless diagnostic procedure may result in peritonsillar infection.

Since *focal infection* (p 42) very commonly originates in tonsillar tissue the importance of the examination of the tonsil cannot be over emphasized. The size of the tonsil is relatively unimportant. Large tonsils that virtually meet in the median line may be free from significant infection. Enlarged tonsils often contain deep *crypts* filled with inspissated material. While the appearance is ugly this does not necessarily indicate disease.

The most seriously infected tonsils are often those with an innocent smooth surface. These may be deeply buried but after removal and sectioning it is found that the entire structure has been replaced by abscess cavities.

The tonsil is frequently the site of acute infection (p 2154). In simple tonsillitis produced by the common respiratory invaders there may be present exudate which is readily wiped away. Membranous inflammation with an adherent exudate suggests the possibility of invasion by the *diphtheria bacillus* (p 302). Very delicate white patches may contain the spirochete of syphilis. Suspicious lesions require *darkfield examination* (p 45). Tonsillar exudates should be examined by spread and culture (p 52).

Ulceration or gangrene of the tonsil (p 2155) requires laboratory investigation. While this type of inflammation may occur as a manifestation of local infection with the Vincent organisms it usually is associated with a underlying *blood dyscrasia* (p 1085). Ulcers of the tonsil may also be produced in *primary syphilis* (p 3278), *tuberculosis* (p 252) and *malignancy*. A notoriously ominous angina is present in the *agranulocytosis* (p 2070) which commonly results from the use of amidopyrine, arspenamines or the sulfonamides.

Peritonsillar abscess (quinsy) is characterized by the inability of the patient to open the mouth widely and the projection anteriorly of the involved tonsil. There is usually marked edema of the uvula.

See *Differential Diagnosis of Pain in the Throat* (p 2071), *Hoarseness* (p 2160), *Nasal Discharge* (p 2100), *Epistaxis* (p 2123), *Dysphagia and Pain on Swallowing* (p 1723), *Cough* (p 2050), *Hemoptysis* (p 2058), *Sneezing* (p 2064), *Tinnitus* (p 2141), *Cryptogenic Fever* (pp 26, 2760), *Stridor and Stertor* (pp 2166, 2732).

THE LARYNX

The larynx is readily examined by *indirect mirror laryngoscopy* but direct examination requires the services of the specialist (p 2036).

ANATOMIC REVIEW

The larynx opens widely into the hypopharynx above while below it is continuous with the subglottic space and the trachea. It is a cartilaginous box lying below the hyoid bone and the base of the tongue. It is bounded on either side by the carotid sheath and is separated from the vertebral column posteriorly by the hypopharynx, the upper part of the esophagus and the prevertebral muscles and fascia. In the midline it lies just beneath the skin where the prominence of the thyroid cartilage is known as the "Adam's apple." Laterally it is covered by the neck muscles and the lobes of the thyroid gland.

The *laryngeal skeleton* is composed of the several cartilages joined by membranous tissues through one of which the thyrohyoid membrane, the superior laryngeal nerve and artery pass to gain the interior of the larynx. The lower margin of the thyroid cartilage is attached to the upper margin of the cricoid by the cricothyroid membrane, the central portion of which is the *conus elasticus* to which the true vocal cords are attached anteriorly. The vocal cords are attached posteriorly to the vocal processes of the arytenoid cartilages. The true vocal cords are triangular on cross section with a flat upper surface. The free margin is thin and sharp and its epithelium is adherent to the underlying *conus elasticus* lateral to which is the *thyro-arytenoid muscle* which forms the major substance of the cord. The glottis, the space between the free margins of the true vocal cords, is the narrowest portion of the larynx.

Below the true cords is the *subglottic space* which is narrower above and widened below to become the lumen of the trachea. Between the true and the false cords are situated the *laryngeal ventricles*. The *laryngeal vestibule* includes that part of the larynx from its upper margin to the margins of the false cords.

The *epiglottis* is attached to the thyroid cartilage just above the vocal cord to the tongue by the median *glosso-epiglottic fold* and laterally to the walls of the pharynx by the *pharyngo-epiglottic folds*. Between these folds lies the *collecua*.

The *laryngeal cavity* is formed in front by the epiglottis and laterally by the *aryepiglottic fold* which contains a small mound containing the *corniculate and cuneiform cartilages* on the tip of the arytenoid space. Lateral to these *aryepiglottic folds* is the *pyriform recess*. The *laryngeal mucosa* is densely adherent over the vocal cords and the *laryngeal surface* of the epiglottis. Elsewhere it is loosely attached and hence subject to marked swelling and inflammation.

The Laryngeal Musculature—The *extrinsic muscles* of the larynx are the *thyrohyoid*, the *sternohyoid* and the *omohyoid*. The *intrinsic muscles* are grouped according to their functions. The *adductors* of the vocal cords are the *lateral crico-arytenoid*, the *thyro-arytenoid* and the *interarytenoid muscles*. The *dilator* is principally the *posterior crico-arytenoid muscle* which is the real abductor of the vocal cords. The *tensors* of the vocal cords are the *cricothyroid* and the *thyro-arytenoid muscles*.

The Innervation of the Larynx—With the exception of the cricothyroid, the laryngeal muscles are innervated by the recurrent laryngeal branch of the vagus nerve. The cricothyroid muscle is supplied by the external branch of the superior laryngeal nerve while the interarytenoid is sometimes similarly supplied by the superior laryngeal nerve. The inferior laryngeal or recurrent nerve enters the larynx from below and runs into the groove between the esophagus and the trachea.

The Vessels of the Larynx—The blood supply of the larynx is through the *superior laryngeal artery*, a branch of the superior thyroid division of the external carotid artery. The *inferior laryngeal artery*, a branch of the inferior thyroid artery, enters the larynx with the recurrent nerve from below.

Function of the Larynx—The larynx is an air passage whose patency is maintained by its cartilaginous walls. The glottis is the narrowest portion of the lumen. It is open during inspiration and expiration and phonation but closes during coughing, swallowing and vomiting.

The reflex cough originating in the larynx prevents the entrance of foreign bodies into the trachea and bronch. The mucosa of the vocal cords and ventricular band is very sensitive so that contact with a foreign body immediately provokes a powerful blast of air to remove the irritant.

The production of voice is the most important function of the larynx. The passage of a current of air from the lungs during inspiration against the thin margins and subglottic surfaces of the true vocal cords when they are tense and approximated sends them into vibration. The waves pass out through the mouth, pharynx and nose with the expired air.

The larynx and the trachea serve for the removal of drainage from the bronchi and lungs, the secretion being brought up by the action of the cilia of the trachea and bronchi aided by the cough reflex.

EXAMINATION OF THE LARYNX

The larynx is examined by *direct* or *indirect laryngoscopy*. The former requires expert technique (p. 2036).

Technic of Indirect Laryngoscopy—The technic of mirror examination of the larynx, hypopharynx and upper trachea should not be difficult if care

DIFFERENTIAL DIAGNOSIS OF

*Abnormalities of the Larynx
Observed by Endoscopy*

Disturbances of the larynx are specialist province almost without exception. It requires expert diagnostic investigation and therapeutic management to deal with the chronic infections and neoplasms. The practitioner must content himself with the visualization of the larynx so as to make early reference to his colleague in the event that any abnormality is noted.

Lesions of the larynx invariably are associated with hoarseness, dysphonia and disturbances in voice production. The present chart therefore refers to these subjective symptoms as well as to the objective abnormalities that are observed.

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|--------------------------------|--|
| Congenital Anomalies | Laryngismus stridulus with inspiratory crow (p 3732) Webbed diaphragm and diverticula of larynx demonstrable by endoscopy (p 3607) Refer to laryngologist. |
| Physical and Chemical Injuries | Misuse of voice producing hemorrhage of cord, pachylaryngitis and singer's nodes (p 2046) Burns particularly from lye with later laryngeal stenosis (p 2046) Impaction of foreign bodies (p 2046) Refer to specialist. |
| Metabolic Disorders | Angioneurotic edema with impairment of air way (p 2101) Note effects of an epinephrine spray and of scarification. Prepare for tracheotomy if necessary (p 3957) Acromegaly of cords. Note low basal metabolic rate and attempt therapeutic result with thyroid extract (p 1189) |
| Neurogenic Disturbances | Hysterical aphonia (p 2691) Unilateral or bilateral paralysis of superior or inferior recurrent laryngeal nerves (p 2091) Tetes of cord (p 1464) Obtain specialist consultation. |
| Acute Inflammations | Acute laryngitis associated with upper respiratory infection (p 2159) Membranous laryngitis due to diphtheria. Demonstrate <i>C. diphtheriae</i> by smear and culture. Supplement antibiotic therapy with antitoxin (p 302) Secondary syphilis with mucous patches. Get darkfield microscopy and Wassermann reaction. Start intensive antibiotic therapy (p 3280) Acute nonspecific perichondritis and chondritis (p 2159) Laryngeal abscess (p 2164) Refer to specialist. |
| Chronic Inflammations | Hypertrophic and atrophic laryngitis (p 2161) Polypoid and cystic laryngitis. Chronic ulcerative laryngitis. Suspect syphilis and tuberculosis. Get darkfield microscopy (p 45) spread for tubercle bacilli and biopsy (p 3935) |
| Neoplasm | Papilloma and carcinoma (p 2072) Refer to laryngologist for biopsy (p 3935) |

is taken and the patient is relaxed. The patient is seated with the head tilted slightly upward and the chest forward. He breathes quietly with the mouth opened widely. The polished laryngeal mirror is held in the examiner's right hand and is warmed over an alcohol lamp with the mirror side against the flame. It is tested for comfortable heat against the palm of the hand and should be warm enough to prevent clouding from the patient's breath and not too warm to be uncomfortable.

The tongue is protruded and seized lightly with a piece of muslin between forefinger and thumb of the left hand. The thumb should be on the upper surface. The tongue is gently drawn out of the mouth without pulling the frenulum against the lower teeth. The mirror is inserted into the mouth with the reflecting surface down and passed above the tongue without touching it. The soft palate and uvula are pressed posteriorly against the posterior pharyngeal wall with the back of the mirror. Meanwhile the patient is asked to breathe quietly and deeply without gagging or coughing.

When the beam of light from the head mirror has been concentrated on the laryngeal mirror the view of the structures of the base of the tongue, the larynx, hypopharynx and trachea is best obtained when the patient phonates EEEEEEE. If the patient cannot be made to relax a spray with 0.5 per cent pantocain against the soft palate and the base of the tongue suffices to overcome gagging and retching.

Indirect laryngoscopy can often be accomplished in young children if their confidence is gained and they cooperate. When this is not feasible *direct laryngoscopy* (p. 2025) has to be resorted to for a proper inspection of the larynx.

Mirror Image by Indirect Laryngoscopy—In the laryngeal mirror the image is inverted, anterior structures appearing posterior and vice versa. At the base of the tongue the foramen cecum, the vallecula and the lingual tonsil are sought. There should not be sufficient lymphoid tissue to fill up the space between the base of the tongue and the anterior surface of the epiglottis. Inflammation of the lingual tonsil, which often hypertrophies after faucial tonsillectomy, is a frequent cause of *chronic cough* (p. 2050).

The *epiglottis* is recognized behind the tongue and in front of the superior opening of the larynx. It is a thin, flexible lamella of yellowish fibrocartilage. It functions as the lid which covers the superior opening of the larynx, preventing the entrance of food particles and fluids into the air passages. Behind the superior aperture of the larynx reveals the projection of the apices of the *arytenoid cartilages*. The *aryteno-epiglottic fold* extends from these cartilages behind to the epiglottis in front. The fold is marked by the whitish prominence that indicates the position of the *cuneiform cartilage*.

The *superior or false vocal cords* and the *inferior or true vocal cords* span the superior aperture of the larynx from behind forward. The cords are pearly white, glistening, smooth and regular. Between the true vocal cords is a narrow, triangular fissure, the *rima glottidis*. The subglottic space and the rings of the trachea are seen below the cords. Between the true and false cords is the laryngeal ventricle and above the false cords the laryngeal vestibule.

Deviations from the normal appearance of the cords, *edema in the*

 DIFFERENTIAL DIAGNOSIS OF

Visible Abnormalities of the Canal and Drum of the Ear

The external auditory canal is examined readily with electrodiagnostic equipment. Visual examination of the tympanic membrane is routine in the survey of the infant. In case ear infections are of great frequency and the examiner lacks subjective direction to the site of the lesion (p 2731) It is of particular importance to differentiate infections involving the drum from those of the middle ear. In myringitis the bulla forms on the surface and the inflammatory process rarely extends to the middle ear unless a paracentesis is done inadvertently. With a true otitis the middle ear is involved and the bulging of the drum is an indication for paracentesis to obtain drainage. When in doubt the practitioner should consult the specialist otologist.

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|---|--|
| Cerumen | May cause itching, pain and impairment of hearing (p 2019) |
| Cholesteatoma | Deposition of grumous mass at margin of perforation. Refer to otologist for x rays and diagnostic survey (p 2015) |
| Perforation of Drum | Following paracentesis (p 2151) As a sequel to otitis media (p 2144) Penetration by foreign body. From violent compression as the result of diving, altitude flying, power dives, explosions, blows over the ear, or forceful dilatation of the eustachian tubes. With fractures of the temporal bone involving the cavity of the middle ear (p 1450) Note bloody discharge (p 2150) Obtain mastoid x rays for immediate information and for later comparison. |
| Retraction of Drum | Prominence of ossicles with loss of light reflex (p 3610) Usually due to obstruction of eustachian tube. May be associated with tinnitus and diminution in hearing (p 2019) |
| Foreign Body | Asymptomatic or associated with itching, impairment of hearing and pain. Insect requires careful extraction by specialist. |
| Furunculosis of External Auditory Canal | Intensely painful pyoderma (p 2111) May cause wide spread edema with obstruction of canal and extension over mastoid process and temporal bone (p 2146) Use local and systemic antibiotic therapy (p 106) |
| Herpes Zoster | Vesicular eruption with involvement of canal and drum. Intense pain with similar lesion along external course of nerve (p 435) |
| Mastoiditis | Following otitis media (p 2146) Look for drooping of postero-superior quadrant of drum, persistence of discharge or sudden cessation of discharge. Tenderness over mastoid tip, antrum, zygoma or digastric triangle (p 3611) Get comparative x rays and refer to otologist. Start intensive anti-infective therapy. |
| Acute Myringitis | Bullous infection of drum without involvement of middle ear. Usually secondary to upper respiratory infection (p 2143) Intense pain, diminution in hearing and only minor constitutional manifestations (p 2146) Avoid paracentesis. Consider antibiotic therapy with penicillin (p 106) |

CONTINUED

Osteoma and Hyperostoses of Canal

Firm bony tumors with obstruction of canal and impaction of cerumen. May cause it hing pain and impairment of hearing. Refer to otologist.

Acute Otitis Media

Usually a complication of upper respiratory infection or exanthematous diseases. Fulness and pain in ear, diminution of hearing, constitutional manifestations and bulging of drum with later perforation (p. 2144). Institute anti-infective treatment but consult otologist for paracentesis and observation.

Chronic Otitis Media

Persistent discharge with tinnitus, head noises, fulness in ear and increasing impairment of hearing. May be associated with perforation or retraction (p. 2151). Refer to otologist for x-rays and diagnostic observation.

Otomycois

Eczema of external auditory canal with itching, pain, impaction of cerumen and frequent attacks of furunculosis (p. 2111). Attempt to isolate fungus. Treat locally with mycomides or radium (p. 3128).

subglottic space the presence of *exudate*, *ulceration* or *neoplasm* demand expert consultation. The laryngologist by direct laryngoscopy obtains a better view and specimens for examination by dark field, bacteriological cultures and stains and biopsy.

The movements of the cord are noted. The glottis opens during inspiration and closes during expiration. The phonation of EEEE causes the cords to approximate closely. With superior laryngeal paralysis the cricothyroids are unable to tense the cords. When the recurrent laryngeals are involved the abductors and later the adductors fail to function. In the latter instance usually unilateral the affected cord is in the cadaveric position. It remains immobile during respiration and fails to abduct on deep inspiration. Other types of *laryngeal paralysis* are rare and should be referred to the specialist.

See *Differential Diagnosis of Disorders of Speech* (p. 3586) *Cough* (p. 2050) *Hemoptysis* (p. 2058) *Pain in the Throat* (p. 2071) *Hoarseness* (p. 2160) *Stridor and Stertor* (pp. 2166-2732).

THE EAR**THE EXTERNAL EAR**

The external ear is made up of the fibrocartilaginous *auricles* and the *external auditory canal*. The auricles rarely present significant findings. Small *tophi* consisting of uric acid concretions may be found in the helix of the gouty. *Sebaceous cysts* occur in or behind the lobe. *Traumatic chondritis* producing the so-called *cauliflower ear* is characteristically seen in prize fighters and wrestlers.

THE AUDITORY CANAL

The auditory canal commonly contains a certain amount of cerumen or inspissated soap. Not infrequently the mass completely blocks the canal and must be removed before it is possible to see the drum. Occa-

sionally a crust is formed which can be lifted away with a small curette. The canal can then be cleansed by means of warm water forcibly ejected from a hand syringe.

Individuals whose auditory canals are found packed with cerumen or soap should be warned to avoid the use of soap in cleansing the ear. Mothers should be taught that the use of the twirled end of a washcloth

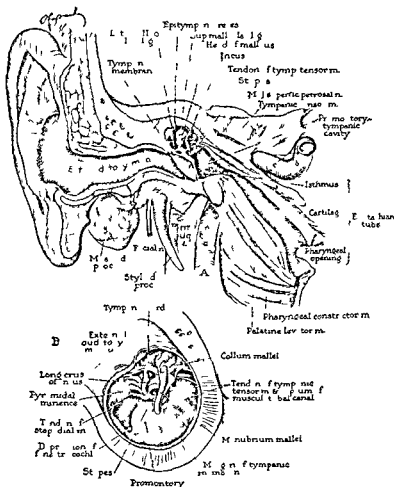


Fig 1048—A Somewhat diagrammatic sectional view of external and middle ear B External view of the tympanic cavity *

serves to push wax in rather than remove it. Men should be cautioned against depositing shaving soap in the meatus while lathering the face.

THE EAR DRUM

The drum or *membrana tympani* (Fig 1048) separates the cavity of the tympanum from the bottom of the external auditory canal. It is a thin semitransparent membrane nearly oval in form and directed obliquely downward and inward so as to form an angle of about 55 degrees with the floor of the canal. The greater part of the circumference of the drum

* Jones and Shepard Manual of Surgical Anatomy

is thickened and fixed in a groove at the inner extremity of the external meatus. The groove is deficient superiorly where two folds (the anterior and posterior malleolar membranes) are prolonged to the short process of the *malleus*. A small triangular portion of membrane situated between these fold is thin and lax. It is termed *Shrapnell's membrane*. It is at this site that the bulging of the drum may be first observed. The remainder of the drum is tightly stretched and is called the tense portion.

Superiorly just below *Shrapnell's membrane* the handle of the *malleus* forms a distinct prominence from which the malleolar folds are given forth anteriorly and posteriorly. From the malleolar prominence the handle extends towards the center of the drum or the *umbo*, the ossicle being placed nearer the anterior than the posterior surface of the drum. The drum is drawn inward towards the tympanic cavity. The most depressed part of the cavity is the *umbo* itself.

Examined by *artificial illumination* the drum membrane should be of pearly gray color and of shiny luster sufficient to reflect a cone of light whose base is anterior and whose apex is at the *umbo*. Here the cone of light meets with the lower portion of the handle of the *malleus* which can then be traced upward toward the malleolar prominence where *Shrapnell's membrane* can be identified above bounded anteriorly and posteriorly by the malleolar folds.

The Auditory Nerve—The auditory nerve is the special pathway for hearing and equilibrium. The field of otology is in general so specialized and its disturbances may cause changes so profound that the practitioner should not deal with lesions of the auditory apparatus without consultation with the specialist (p 2015).

THE MASTOID PROCESS

The mastoid process is palpated behind the ear lobe. Tenderness and edema of the tip speak for infection of the cells.

Tests of the Acuity of Hearing—Audiometers are employed by the otological specialist to test the intensity and range of hearing. These instruments are not recommended for the general practitioner.

Hearing may be roughly tested however by the spoken and whispered word and the ticking of a watch. The patient is instructed to close the eyes and stopper one ear with the finger. Numbers are spoken or whispered by the practitioner who varies the intensity of his voice and the distance between himself and the patient. In a relatively quiet room of average size a reasonably soft whisper should be readily audible. After testing one ear the other is investigated and a comparison is made between the acuity of hearing in the two ears.

A more objective test may be made with an ordinary watch. With eyes closed and one ear stoppered the patient indicates when the watch tick is observed. The distance is measured. The other ear is compared and then both ears are compared with the acuity of hearing of the physician himself who serves as a control.

Vestibular tests for equilibrium are the province of the specialist (p 2015).

See *Differential Diagnosis of Deafness* (p 2019) *Vertigo* (p 2020) *Cough* (p 2050) *Hyperacusis* (p 2096) *Dermatoses of Ear* (p 2113).

Tinnitus (p 2141) *Pain in Ear* (p 2143) *Bleeding and Discharge from Ear* (p 2150) *Cryptogenic Fever in Infancy* (p 2760) *Convulsions in Infancy* (p 2780), *Otogenic Brain Abscess* (p 2148), *Otogenic Meningitis* (p 2148) *Headache* (p 1512) *Sinus Thrombosis* (p 2130)

THE EYE

ANATOMIC REVIEW

Vision is produced by the effects of light waves which cause a photochemical change in the retina. Stimuli, electrical or otherwise are initiated and transmitted to the brain. The sentient layer the retina is part of the central nervous system being connected to the brain by the optic nerves. The whole structure of the eye is designed to protect and nourish the retina and insure the accurate focussing upon it of light rays. The adnexa of the eye include the lids, conjunctivae and lacrimal apparatus.

The Ocular Adnexa *The Eyelids*—The lids (palpebrae) are folds of the external skin which envelop and protect the eyeball. The *palpebral opening* or fissure is the space between the margins of the two lids and the extremities of the aperture are the angles or *canthi*. The free margin of the lid with its rounded anterior border continuous with the skin contains the cilia or eyelashes which arise in two or three irregular rows. The associated sebaceous glands are the *glands of Zeis*. Between the cilia are the *sweat glands of Moll*. The posterior border or lip of the lid margin is sharp. It contains the openings of the *meibomian glands* in front of which dividing the lid into two parts is the gray line or *intermarginal space*.

The *arteries of the eyelids* are derived from ophthalmic and facial trunks. The medial and lateral palpebral arteries anastomose to form two arterial arcades in the upper lid. The lower or marginal runs 3 mm. above the lid margin deep to the orbicularis. The upper or peripheral (retrotarsal) runs along the curved border of the tarsus. In the lower lid only one arcade is present. The *veins* have arcades similar to the arteries. Their drainage is superficial into the anterior facial and superficial temporal veins and deep into the orbital veins going to the cavernous sinus and the pterygoid plexus. The deep drainage is very important in the spread of infection from the lid. The *lymphatics* drain into the pre-auricular, submandibular and parotid lymph glands. The ophthalmic division of the fifth nerve supplies the upper lid and the medial and lateral extremities of both lids while the maxillary division supplies the central part of the lower lid. There is however some overlapping.

The Conjunctiva—The conjunctiva is the mucous membrane which lines the eyelids and is reflected onto the eyeball to form the conjunctival sac. The conjunctiva is divided into four parts. The *palpebral conjunctiva* covers the under surface of the lids and is very adherent to the tarsus. The *conjunctiva of the fornix* is the fold lining the cul-de-sac resulting from the reflection of the conjunctiva from lid to globe. It is loose facilitating ocular motion. The *bulbar conjunctiva* which covers the anterior part of the eyeball is thin and transparent, while the semilunar fold or *plica semilunaris* is a crescentic fold situated near the medial canthus. A vestigial structure in man it represents the third eyelid or nictitating membrane or lower animals.

The *epithelium* of the conjunctiva is columnar in type and is two or three layers thick. The substantia propria consists of a superficial adenoid layer absent at birth it contains many lymphocytes but no true follicles. The deeper fibrous layer contains the vessels and nerves of the conjunctiva. In the bulbar conjunctiva, goblet cells secreting mucus are most numerous. The epithelium in this region is directly continuous with that of the cornea.

The *blood supply* of the conjunctiva is derived from the retrotarsal arcade of the lid whose posterior conjunctival branches anastomose with the anterior ciliary arteries to form a pericorneal plexus. This is the anatomical basis for the conjunctival and ciliary or circumcorneal types of injection. In the former case the congestion is derived from the posterior conjunctival vessels in the latter from the anterior ciliary vessels. The *nerve supply* of the conjunctiva is from the fifth nerve.

The Caruncle—The caruncle is a small red fleshy body lying on the base of the semilunar fold. It represents part of the margin of the lower lid which has been cut off by the development of the inferior lacrimal canaliculus. It is absent in animals which lack a lacrimal apparatus.

The Lacrimal Apparatus—The lacrimal apparatus consists of (1) the lacrimal glands, which are derived from the conjunctiva and secrete tears into the conjunctival sac; (2) the lacrimal passages which conduct tears into the inferior meatus of the nose.

THE LACRIMAL GLAND—The lacrimal gland lies in the upper and outer corner of the orbit in the lacrimal fossa. It is divided into a *superior* (orbital) lobe and an *inferior* (palpebral) lobe also known as the accessory lacrimal gland. The ducts of both lobes, two to five from the superior and six to eight from the inferior lobe, empty into the fornix. The ducts from the superior lobe pass through the inferior lobe, hence extirpation of the latter is functionally equivalent to removal of the whole gland.

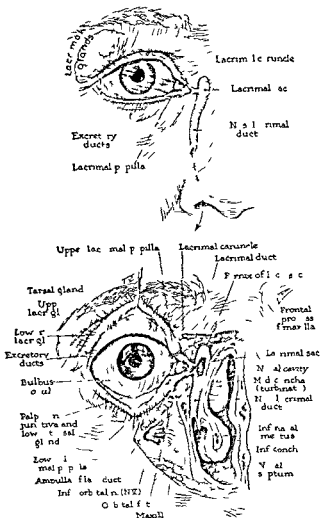


Fig 1049.—The lacrimal apparatus. Upper figure shows surface projection; lower figure shows the general topography with front of nose removed. Note tarsal glands.

The glandular structure resembles that of the salivary glands, containing acini with cuboidal cells. Scattered collections of glands (Krause) of similar character occur throughout upper and lower fornices. Other rudiments occur in the plica and the caruncle.

THE LACRIMAL PASSAGES—The lacrimal passages begin with the *lacrimal puncta*, which are small openings situated in elevations on the margins of the upper and lower lids near the medial extremity. The puncta lead into the *lacrimal canaliculi*, narrow tubular passages

directed vertically and then turning at right angles to proceed horizontally and medially for 8 to 9 mm to empty into the *lacrimal sac*. The ducts may open separately or form a short common trunk.

The *lacrimal sac* lies in the *lacrimal fossa*, the hollow formed by the frontal process of the maxilla anteriorly and the lacrimal bone posteriorly. It is continued downward as the *naso-lacrimal duct* which traverses the canal formed by the lacrimal, maxillary and inferior turbinate bones, emptying into the inferior meatus of the nose. The beginning of the bony canal is the narrowest part and is frequently the site of an obstruction. The duct is about 18 mm long and 4 mm in diameter.

Both the sac and the duct are lined with a double layer of columnar epithelium. Cilia are usually absent in adults. The *substantia propria* consists of adenoid tissue superficially and elastic fibers with many veins deep to it.

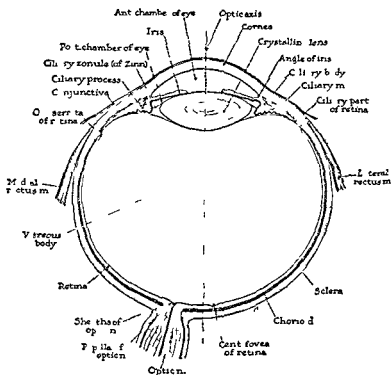


Fig 1050—Section through right eye in horizontal plane magnified about 3 times

The Fibrous Tunic: The Sclera—The sclerotic coat is strong, opaque and inelastic. It is white in color and its outer surface is covered by Tenon's capsule (bulbar fascia) to which it is connected by loose connective tissue traversing the episcleral space. Its thickness varies from 0.3 mm at the insertions of the rectus tendons to 1.0 mm posteriorly. The sclera is pierced anteriorly and posteriorly by numerous vessels and nerves. At the minute perforations are pigmented spots resulting from the migration of chromatophore cells from the perichoroidal space. Although the fibrous tunic is structurally continuous, two large foramina are described in the sclera. The anterior scleral foramen marks the merging of the irregular scleral fibers into the regularly arranged lamellae of the cornea. The posterior scleral foramen allows the exit of the optic nerve.

The sclera is divided into three layers. The *episcleral tissue* consists of loose fibrous and elastic tissue. The *sclera proper* is a dense mass of fibrous connective tissue arranged in criss-crossing bundles, most of which consist of collagenous fibers with numerous interspersed elastic fibers. In the *lamina fusca* or dark layer, the bundles are smaller and contain more elastic fibers. A brown color is imparted to this layer by the presence of a large number of chromato-

phores or pigment cells. Lining the perichoroidal space is a layer of endothelial cells. The few blood vessels present are mainly confined to the episcleral tissues. No lymphatics are present.

The Cornea—The cornea is a clear transparent tissue with a smooth and brilliant surface. It is curved and fits into the anterior scleral foramen with a bevelled margin. By slit lamp inspection its estimated thickness is 0.6 mm. to 0.65 mm.

The cornea is divided into the cornea proper and the limbus, the transition zone between the cornea and the sclera. The cornea proper is composed of five layers. The epithelial layer is of uniform thickness and regularly structurally continuous with the conjunctiva and easily detachable from Bowman's membrane. It is of the stratified pavement type and regenerates very rapidly after injury leaving no opacity. Bowman's membrane or the anterior elastic lamina is a thin clear uniform structureless tissue ending abruptly at the limbus. It is tough and resistant to trauma but it never regenerates and leaves an opacity if destroyed. The substantia propria (corneal stroma) constitutes about 90 per cent of the corneal thickness. It consists of bands of fibres arranged in criss-crossing lamellae running parallel to the surface and connective tissue cells known as corneal corpuscles found in a communitative system of spaces or lacunae between the lamellae. Descemet's membrane is a homogeneous thin elastic layer between the substantia propria and the endothelium readily separable from both. It is resistant to inflammation and readily regenerates. The lining endothelium is a single layer of flat hexagonal cells.

The limbus is a transition zone 1 mm. wide between the cornea and the sclera. Only three layers are present, since Bowman's membrane stops short and Descemet's membrane changes to participate in the formation of the meshwork of the angle of the iris. This region is characterized by the presence of blood vessels and lymphatics both of which are absent in the cornea proper.

The cornea is devoid of blood vessels. The anterior ciliary vessels liberally supply the limbic region and form a circular marginal plexus and the lymphatics are similarly arranged. The cornea proper is richly supplied with nerves derived from the ciliary division of the fifth nerve. These are essentially nerves of pain.

The Vascular Tunic—The vascular tunic presents from behind forward (1) the choroid (2) the ciliary body and (3) the iris. These subdivisions are so intimately related that usually when one becomes inflamed the others also are involved.

The Choroid—The choroid is made up of blood vessels united by connective tissue containing many pigmented cells. It extends from the ora serrata to the posterior scleral foramen and varies in thickness from 0.1 mm. anteriorly to 0.2 mm. posteriorly. A potential space the perichoroidal space separates it from the sclera, from which it is easily detached. On the other hand on its internal aspect, it is intimately adherent to the pigment epithelium of the retina.

From without inward the choroidal layers consist of (1) the epichoroid (suprachoroid) (2) the vascular layer consisting of large and small vessels (3) the choriocapillaris and (4) the lamina vitrea (Bruch's membrane). The epichoroid is a loose connective tissue containing pigment cells. The vascular layer comprises larger vessels mainly veins which anastomose to form the four vortex veins which emerge from the eye at the inner layers of the retina. The lamina vitrea is composed of an outer elastic and an inner homogeneous layer derived from the retinal pigment epithelium.

The elements of the choroid are transparent except those containing pigment upon which depends the ophthalmoscopic appearance of the fundus.

The Ciliary Body—The ciliary body is that 5 to 6 mm. of the uvea extending from the ora serrata posteriorly to the corneal junction anteriorly. In longitudinal section it is triangular in shape. Its narrow base which is directed anteriorly gives off the root of the iris and its apex is continuous with the choroid. The outer part of the triangle is made up of the ciliary muscles while the external surface next to the sclera is smooth. The inner surface is folded into an anterior part which consists of the radiating ciliary processes or ciliary ciliaris and a posterior oblique ciliary which is broad and flat.

The ciliary body may be considered a continuation forward of the choroid and retina. The ciliary muscle corresponds to the epichoroid. The muscle of accommodation consists of smooth muscle fibres arranged in meridional radial and circular directions. The first two sets are attached to the sclera. In hyperopia the ciliary muscle will be relaxed while in myopia it is poorly developed.

The ciliary processes consist of about seventy-five folds arranged meridionally. Viewed from behind they seem to form a circle. They are extremely vascular the vessels being a continuation of the vessel layer of the choroid. Two layers of epithelium cover the processes

Externally is the pigment epithelium from the retina and internally next to the vitreous a layer of cylindrical nonpigmented cells the *pars optica retinae*. The ciliary processes function in the formation of the intra-ocular fluid. Attached to the processes are the zonular fibers that support the lens.

The Iris—The iris is a colored membrane circular in form hanging behind the cornea immediately in front of the lens separating the anterior from the posterior chamber. The pupil is its central circular opening of variable size which serves to regulate the amount of light entering the eye and helps sharpen retinal images by eliminating marginal rays. Peripherally the iris is attached to the ciliary body by its root the weakest part of the uveal tract. Its anterior surface is irregular with elevations and depressions (crypts). Its topography as well as its color varies with individuals. The posterior surface of the iris is smooth and black broken only by relatively shallow furrows.

The iris consists of a delicate spongy connective tissue stroma containing pigmented cells muscle fibers many nerves and blood vessels. It may be divided into several layers. The endothelium is continuous with that of the cornea and is absent over the crypts. The vessel layer is made up of numerous blood vessels originating from the greater arterial circle of the iris situated in the anterior part of the ciliary body. The stroma in which the vessels lie contain predominantly large branched chromatophores full of brown pigment.

The sphincter and dilator muscles of the iris are of epithelial origin being derived from the anterior prolongation of the pigment layer of the retina. The sphincter is an annular band 0.8 mm wide encircling the pupillary border. It is supplied by the third nerve and contracts the pupil. The dilator muscle is a uniform layer in the posterior part of the iris between the stroma and the pigment epithelium. It extends over the entire area of the iris with the exception of the tip. It consists of long spindle shaped cells arranged meridionally and retains more of an epithelial character than the sphincter.

The pigment epithelium of the iris forms the posterior surface and is a deeply pigmented layer of cells. It is a continuation of the ciliary epithelium. The internal limiting membrane is continuous with the same structure posteriorly.

The color of the iris depends on the amount of pigment in the stroma which varies in individuals and on the pigment epithelium which is always heavily pigmented. At birth in the white races the iris appears blue because of a paucity of stroma pigment whereas in the dark races the stroma is heavily pigmented by that time. The color turns to gray or brown after birth according to the amount of pigment later developed in the stroma.

The Chambers of the Eye *The Anterior Chamber*—The anterior chamber is the space between the posterior surface of the cornea and the anterior surface of the iris and lens. The peripheral angle of the chamber is known as the filtration angle of the eye. Except for the area over the lens and the crypts of the iris the entire anterior chamber is lined with endothelium. Over the crypts the aqueous or the fluid of the chamber is in direct contact with the iris stroma and blood vessels. The depth of the chamber is greatest at the center where it averages 3.5 mm. It is shallow in infants and in old age. In hyperopia it is shallower than in myopia and in accommodation shallower than when the eye is at rest.

Physiologically the most important part of the anterior chamber is the angle. Since the iris inserts into the base of the ciliary body instead of the corneal limbus a recess in the chamber occurs extending 1 mm beyond the limbus. The outer border of this recess contains the trabeculum a loose meshwork of trabeculae covered by endothelium and is readily permeable to the aqueous.

Immediately external to the trabeculum of the angle lies a venous sinus—the canal of Schlemm. This structure which is not a single channel but is broken irregularly into several compartments encircles the entire eye at this level. It is completely surrounded by sclera except where it is in contact with the loose trabeculum. The aqueous from the anterior chamber filters through into the endothelial lined cavity from which it enters an intricate venous plexus in the surrounding sclera finally leaving the eye through the anterior ciliary veins. Schlemm's canal proper does not contain blood elements except in extreme venous stasis. Contractions of the ciliary muscle seem to open the canal.

The Posterior Chamber—The posterior chamber of the eye is the space bounded in front by the iris and behind by the anterior surface of the lens and the anterior face of the zonule. Like the anterior chamber it is filled by aqueous humor.

The Neural Elements *The Retina*—In the early embryo the optic vesicle which is an outpouching of the brain is invaginated to form the optic cup which has two layers from which the retina is developed. The outer layer forms the thin pigment epithelium and the inner the complicated structure of the *pars optica retinae* or practically the whole retina.

The potential space of the optic vesicle remains throughout life and in a detachment of the retina separation occurs between the two layers the pigment epithelium adhering firmly to the choroid

The pigment epithelium consists of a single layer of hexagonal cells containing melanin pigment whereas the retina proper (*pars optica retinae*) is a delicate transparent membrane running from the papilla to the ora serrata forming the internal tunic of the eyeball. Near the disk it measures about 0.5 mm but thins out toward the periphery to 0.1 mm. A concave depression the *fovea centralis* measuring 2 mm in diameter is situated a little below the center of the optic disk and 3.5 mm temporal from its border. In the center of the fovea centralis is a small concavity called the *foveola*. With the ophthalmoscope a ringlike macular reflex with a dot in the center (foveolar reflex) marks the concavities. Observed grossly the fovea has a yellow color and has been called the *macula lutea*.

Exclusive of the pigment epithelium there are nine component retinal layers beginning with the outer layer they are

| | |
|------------------------------|---------------------------------|
| 1 Layer of rods and cones | } <i>Neuro-epithelial Layer</i> |
| 2 External limiting membrane | |
| 3 Outer nuclear layer | |
| 4 Outer plexiform layer | |
| 5 Inner nuclear layer | } <i>Cerebral Layer</i> |
| 6 Inner plexiform layer | |
| 7 Ganglion cell layer | |
| 8 Nerve fiber layer | |
| 9 Internal limiting membrane | |

Two kinds of tissue are present nervous elements and supporting tissue (Mueller's fibers)

The layer of *rods and cones* is the light perceiving layer and consists of cylindrical structures arranged vertically. The rods are thinner and much more numerous than the cones. They contain a pigment known as visual purple or rhodopsin. At the macula cones predominate whereas in the fovea only cones are present. Here all layers of the retina are thinner and the nerve fiber layer is absent. Scattered supporting tissue also is present in this layer.

The *external limiting membrane* is very thin. It is continuous with Mueller's fibers. The *outer nuclear layer* contains the nuclei of the rods and cones and Mueller's fibers. The *outer plexiform layer* is made up of fibers. Here the synapses occur between the rod and cone fibers and the fibers of the rod and cone bipolar cells of the inner nuclear layer. The latter also contains the nuclei of Mueller's fibers and those of the horizontal and amacrine cells which connect the retinal elements.

The *inner plexiform layer* is composed of fibers and synapses going to the ganglion cell layer while the *ganglion cell layer* (Neuron III) has efferent fibers which travel in the *nerve fiber layer* parallel to the surface to form the optic nerve at the disk. These fibers are non-medullated.

The *internal limiting membrane* is a thin homogeneous hyaloid membrane.

The visual impulse is received by the rods and cones which together with the nuclei and axones constitute the *first neuron*. The *second neuron* is made up of the rod and cone bipolar cells which transmit the impulse to the *third neuron* or the ganglion cells whose axones continue through the optic nerve to the primary optic centers of the brain.

The first neuron is the neuro-epithelial layer which has no blood vessels but gets nourishment by transudation from the choriocapillaries. The second and third neurons known as the *cerebral layer* are supplied by branches of the central retinal artery the larger branches being in the nerve fiber layer. The vessels resemble those in the brain being end arteries with perivascular lymphatic sheaths. Capillaries supply the entire inner nuclear layer but external to this layer no vessels are present. The fovea is capillary free for an area of 0.5 mm in diameter.

The Optic Nerve—The optic nerve runs from the eye to the optic chiasm. It varies in length from 35 to 55 mm. It may be divided into (1) an *intra-ocular portion* or the head of the optic nerve (1 mm long) (2) an *orbital portion* extending from the eyeball to the optic foramen (25 mm long) and (3) an *intracranial portion* from the foramen to the chiasm (10 mm long).

The intra-ocular portion of the optic nerve pierces the choroid and sclera a little to the inner side of the posterior pole of the eyeball. Here the external layers of the sclera are

reflected backwards into the outer sheaths of the nerve while the innermost lamellae continue across the opening to form a perforated diaphragm known as the *lamina cribrosa*. At this point the nerve fibers lose their medullated character and become transparent. The portion of the nerve in front of the lamina cribrosa is called the *head of the optic nerve* (papilla nervi optici; optic disk). It measures about 1.5 mm in diameter and has a funnel shaped depression known as the *physiological cup*.

The lamina cribrosa is the weakest spot of all the tunics of the eye. In glaucoma or increased tension it is the first to give way and form an excavation.

The orbital portion of the optic nerve is 25 mm long and S-shaped so as not to interfere with ocular movements. It consists of the nerve trunk and the sheaths enveloping it. It measures 3 to 4 mm in diameter being thicker than the nerve head due to the myelin sheaths. The nerve trunk consists of perhaps 500,000 nerve fibers arranged in bundles with supporting tissue. There are three sheaths of the nerve which originate from the three enveloping membranes of the brain. From within out these are the pial, arachnoid and dural sheaths. The middle sheath, the arachnoid, divides the *intratraginal space* between the dural and pial sheaths into lymph spaces lined by endothelium. Anteriorly the intratraginal space ends in a cul de sac and the sheaths unite with the sclera.

The central retinal blood vessels enter the orbital portion of the nerve about 7 to 12 mm behind the eyeball and run in the axis of the nerve to the papilla.

The intracranial portion of the optic nerve leaves the orbit through the optic foramen along with the ophthalmic artery. It is short and 4 to 7 mm wide.

The Refractive Media—Normally these media are transparent and are so constructed as to focus light rays accurately upon the retina.

The refractive media of the eye consist of the cornea in front, the aqueous filling the anterior chamber, the lens and the vitreous.

The Aqueous—The aqueous is a watery fluid containing some albumin and salts.

The Lens—The crystalline lens lies between the iris and the vitreous. It is transparent though slightly yellow in color, biconvex with the posterior surface more curved than the anterior. It has an anterior pole, a posterior pole and a rounded circumference, the equator. It measures about 4 mm in width and 9 mm in its equatorial diameter.

The lens is derived from a vesicular invagination of the surface ectoderm, the cells of which secrete a capsule around themselves. Originally a hollow sphere of epithelium cells, its posterior cells elongate to form the primitive lens fibers, thus filling the lens vesicle and forming the *embryonic nucleus*. The anterior epithelium keeps its original character. The cells at the equator retain their ability to form lens fibers and do so throughout life. The fibers grow toward the two poles anteriorly and posteriorly and surround the primitive fibers. Where these fibers meet in front and behind, visible suture lines are formed. Thus a series of layers arises and the lens, being unable to shed tissue as does the skin, becomes larger in size with age.

The lens has a capsule which is thin and homogeneous. Anteriorly it is thicker than behind. It has a single layer of cuboidal epithelium underneath it.

Essentially the lens substance consists of concentric layers of hexagonal fibers which join each other in front and behind to form the two sutures. The anterior is Y shaped and the posterior is an inverted Y. From a practical point of view, the adult lens may be considered to consist of a soft cortex and a harder nucleus. Slit lamp examination reveals that the nucleus consists of a number of layers recording the history of its development. Not until the age of 25 is a distinct nucleus present. With advancing years it increases in size and in very old age almost the entire lens is converted into nucleus or is sclerosed. Since accommodation requires changing of the shape of the lens, it follows that the increasing sclerosis causes diminished accommodative power with advancing age (presbyopia).

The lens has no blood vessels.

The zonule forms the suspensory ligament of the lens. It consists of delicate homogeneous fibers arising from the inner surface of the ciliary body and attaching internally to the lens capsule at the equator and a little in front of and behind it. The individual fibers may be agglutinated together to form an anterior and a posterior sheath with a canal (Hannover) in between. The posterior layer is separate from the anterior face of the vitreous by a potential space (canal of Petit).

The Vitreous—The vitreous is a clear transparent gel of semisolid consistency filling the eyeball behind the lens. The apparent texture seen on slit lamp consisting of a network is claimed to be merely an optical effect. A condensation of its outer surface is known as the hyaloid membrane. In embryonic life the hyaloid artery runs from the optic disk to the back of the lens through the vitreous. After it disappears the *hyaloid canal of Cloquet* or some-

thing that resembles a canal remains. Nourishment of the vitreous is obtained from the surrounding structures as it has no blood vessels.

The Blood Vessels of the Eye The Ciliary Circulation.—The ciliary circulation supplies the whole of the eye except the retina and part of the optic nerve. The *posterior ciliary arteries* arise in several trunks from the ophthalmic artery and divide into about 20 vessels piercing the sclera in a ring around the optic nerve (the short posterior ciliary arteries). Two larger long posterior ciliary arteries enter the eyeball somewhat external to the ring, one on each side of it.

The short posterior ciliary arteries supply (1) most of the posterior half of the choroid (2) the palpebral sheath of the optic nerve and the lamina cribrosa by means of the circle of Zinn.

The two long posterior ciliary arteries (medial and lateral) run forward in the perichoroidal space to the ciliary body where they bifurcate in the muscle to supply it and anastomose with branches of the anterior ciliary arteries to form the *greater arterial circle of the iris*.

The *anterior ciliary arteries* usually seven in number arise from the muscular branches of the ophthalmic artery to the recti muscles. Piercing the sclera at the level of the ciliary body they enter its muscle and anastomose with branches of the long posterior ciliary arteries to form the greater circle of the iris.

The *greater arterial circle of the iris* runs around the anterior part of the ciliary body just behind the root of the iris. It supplies (1) ciliary muscle (2) ciliary processes (3) anterior half of the choroid (4) the iris. A second *lesser circle of the iris* at the collarette is also given off.

The *venous return* of the uveal tract is by the vortex anterior and posterior ciliary veins. Most of the blood from the iris and ciliary body and all of the choroid is collected by the four vortex veins which leave the eye 6 to 8 mm. behind the equator between the recti muscles. The two upper ones enter the superior orbital vein, the two inferior ones the inferior orbital vein. The anterior ciliary veins drain the anterior ciliary musculature and form the *nasolacrimal plexus* associated with the canal of Schlemm and finally enter the orbital veins. The posterior ciliary veins are few, draining the posterior sclera only.

The Retinal Circulation.—The retina is supplied by the central retinal artery which arises from the ophthalmic at the apex of the orbit. It enters the optic nerve a short distance behind the eyeball and enters the eye at the optic disk. Aside from recurrent branches to supply the nerve its main branches are the *superior and inferior papillary arteries*, each of which shortly divides into a *nasal and temporal* branch. The whole retina is uniformly supplied except a area of 0.5 mm. in diameter at the fovea.

The retinal veins in general run with the arteries and emerge into the central retinal vein which usually flows into the cavernous sinus directly. It may however join one of the ophthalmic veins, usually the superior.

As described previously the vessels are confined to the cerebral layers, stopping at the outer border of the inner nuclear layer.

The Nerves of the Eye.—The nerves within the eyeball are ciliary nerves originating from the ciliary ganglion on the nasociliary nerve. From the former arise the 10 to 15 *short ciliary nerves* which have sensory and sympathetic fibers. The *long ciliary nerves* derived from the nasociliary are largely sensory but carry sympathetic pupillo-dilator fibers as well. One large nerve accompanies each of the long posterior ciliary arteries.

The Orbit.—The orbits or eye-sockets are two bony cavities in the skull situated on either side of the root of the nose. The orbit forms a quadrilateral pyramid with the optic foramen as its apex. The base is directed forward and the walls of the orbit become thickened at their anterior margin onto a strong bony margin. The nasal walls of the two orbits are parallel while the two temporal walls make an angle of 45° with the nasal ones and thus an angle of 90° with each other.

In the posterior part of the orbit near its apex are many important apertures. The *opt. foramen* passes between the two roots of the lesser wing of the sphenoid into the middle cranial fossa and transmits the optic nerve, the ophthalmic artery and branches of the sympathetic. The *superior orbital fissure* temporal to this lies at the junction of the upper and lateral walls and is bounded by the lesser and greater wings of the sphenoid. It opens into the middle fossa and transmits the ophthalmic vein, the nerves for the ocular muscles, the third, fourth and sixth nerves, the ophthalmic division of the fifth nerve as well as sympathetic fibers. The *inferior orbital fissure* lies at the junction of the temporal and inferior walls of the orbit between the greater wing of the sphenoid and the superior maxilla. It opens into

the sphenopalatine fossa and transmits the maxillary (second) division of the fifth nerve the infra orbital artery the zygomatic nerve and branches from the sphenopalatine ganglion

The orbit is pierced by six other openings the *sphenoidal fissure* the *sphenomaxillary fissure* the *anterior and posterior ethmoidal foramina* the *malar foramen* and the *canal for the nasal duct*

The orbit possesses a roof floor lateral and medial walls

The *roof* is made up of the orbital plate of the *frontal bone* anteriorly and the lesser wing of the *sphenoid posteriorly* The margin is formed by the *frontal bone* and is interrupted to form the *supra orbital notch* The *lateral wall* consists of the *zygomatic bone* anteriorly and the greater wing of the *sphenoid posteriorly* The orbital margin is strongest laterally and is made up of the *zygomatic process of the frontal bone* above and the *zygoma* below The *floor* is composed of the *orbital plate of the maxilla*, centrally the *zygoma* laterally and the orbital process of the *palatine bone* posteriorly The *infra-orbital margin* consists of the *frontal process of the maxilla*, medially and the *zygomatic bone* laterally

The *medial wall* is made up of four bones The *lamina papyracea* of the *ethmoid* forms the main central part The *angular process of the frontal bone* is above and anterior the *lacrimal bone* below and anterior and the lateral aspect of the body of the *sphenoid* is posterior

The medial aspect of the orbital margin contains the *fossa for the lacrimal sac*

The relations of the orbit are important in the study of extension of infection Superiorly lies the *anterior cranial fossa* the *frontal and supra-orbital sinuses* laterally the *middle cranial fossa* the *temporal fossa* and the *pterygopalatine fossa* inferiorly the *maxillary sinus* and the *palatine air cell* and medially the *ethmoid cells* the *nasal cavity* and the *sphenoidal sinus* (posteriorly) Thus the orbit is in close proximity to the *nasal accessory sinuses* from which infection may spread

The contents of the orbit include the *eyeball* and *optic nerve* the *ocular muscles* the *lacrimal gland* *blood vessels* and *nerves* The *interstices* are filled with *orbital fat* and the *fascia* of the orbit holds the whole structure together

The *orbital fascia* a continuation of the *dura mater* serves as the *periosteum* or *periorbita* At the margin of the orbit it continues to both the *tarsi of the lids* and to the *canthal ligaments* forming the *tarso-orbital fascia* and in effect an *anterior orbital wall* Another layer covers the muscles and sends out connecting processes to the other muscles and the margins of the orbit (*check ligaments*) The *fascia bulbi* or *Tenon's capsule* is a *fibrous capsule* extending anteriorly as far as the *corneoscleral junction* and posteriorly to the *optic nerve* Where the muscles pierce it *Tenon's capsule* is reflected upon the muscles and becomes continuous with the *fascia* covering them

The Orbital Muscles—The orbit has *extrinsic eye muscles* muscles to the lid and *plain muscle*

The *extrinsic muscles of the eye* consist of four *recti* or *straight muscles* and the two *obliques* The *recti* are the *medial or internal* the *lateral or external* the *superior* and *inferior* All arise from the *annulus of Zinn* at the orbital apex The *annulus* encircles the *optic foramen* and the *medial end of the superior orbital fissure* As they run forward they diverge to form a *muscle cone* or *funnel* surrounding the *eyeball* The *recti* insert into the *sclera* in the positions the *r* names indicate the *medial 5.5 mm* from the *limbus* the *inferior 6.5 mm* the *lateral 6.9 mm* and the *superior 7.7 mm*

The *oblique muscles* are the *superior* and the *inferior* The *superior oblique* also arises from the margin of the *optic foramen* and runs forward along the upper and inner wall of the *trochlea* a *fibrous loop or pulley* near the margin of the orbit The muscle after traversing the *trochlea* turns backward downwards and laterally passing beneath the *superior rectus* to insert in the form of a fan into the *sclera* behind the *equator*

The *inferior oblique* arises from the lower margin of the orbit near its medial edge It runs under the *inferior rectus* to the outer side of the *eyeball* where it is inserted into the posterolateral part of the globe behind the *equator*

The *superior rectus* is innervated by the *superior division of the third nerve* The *medial and inferior recti* and the *inferior oblique* are also supplied by the *third nerve* the latter two by its *inferior branch* The *superior oblique* is supplied by the *fourth nerve* while the *lateral rectus* is innervated by the *sixth nerve*

Thus with *third nerve palsy* (p 1645) there is *ptosis* of the lid and the eye cannot move inward upward or downward The *sixth nerve* controls the outward motion hence the *sixth nerve palsy* is characterized by inability to move the eye outward Abnormalities of the ocular movement are known as *strabismus* or *squint* (p 1530) *Strabismus* is *divergent* or

external if the eye turns out and convergent or internal if it turns in. Either type of strabismus may be concomitant when there is no true paralysis or paralytic where there is actual limitation or loss of movement of one or more of the extrinsic ocular muscles.

The muscle of the lid is the *levator palpebrae superioris* which arises at the apex of the orbit above the *anulus* passes forward above the *superior rectus* and terminates in an

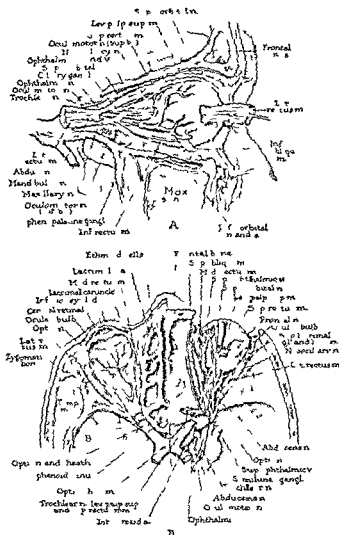


Fig. 1051.—*l*, per figure Dissection exposing the contents of the right orbital cavity lateral view. The lateral rectus muscle has been turned aside. Lower figure Dissection of the orbital cavities viewed from above. The left side represents a horizontal section taken at a somewhat deeper level than on the right.

expanded ponserosus in the upper lid. The insertion is complete involving the skin of the lid, the tarsal plate, the conjunctiva and the orbital margin. It elevates the lid and is supplied by the same superior branch of the third nerve that supplies the superior rectus.

The plan muscle of the orbit includes the oblique muscle of Mueller and the peribulbar

musculature The orbital muscle of Mueller bridges the inferior orbital fissure and joins the inferior oblique anteriorly Posteriorly it runs under the annulus of Zinn The peribulbar musculature is an indefinite group of smooth muscle fibers surrounding the anterior portion of the globe It is closely connected with Tenon's capsule Anteriorly it extends to the lids

Both these muscles are nonstriated and innervated by the sympathetic Theories concerning exophthalmus have involved their overaction

The Blood Vessels of the Orbit—The blood vessels of the orbit with one exception arise from the ophthalmic artery a branch of the internal carotid They include the central retinal artery the posterior ciliary arteries the supra-orbital the palpebral and ethmoidal branches The infra-orbital artery a branch of the internal maxillary supplying minor branches to the lower contents of the orbit, is the exception

The superior ophthalmic vein the largest vein in the orbit empties into the cavernous sinus The inferior ophthalmic vein also does so but a branch goes downwards to the pterygoid plexus Other veins go anteriorly to empty into the facial system

The Nerves of the Orbit—The nerves of the orbit are motor sensory and sympathetic The motor elements are the third fourth and sixth nerves to the extra-ocular muscles and the levator Sensation travels along the ophthalmic or first division of the fifth nerve which enters the orbit through the superior orbital fissure as lacrimal frontal and nasociliary divisions These supply the eyeball lacrimal gland conjunctiva upper lid and surrounding skin



Fig 1052—Technic of focal illumination

The maxillary or second division of the fifth nerve enters as the infra-orbital nerve through the inferior orbital fissure and emerges on the face through the infra-orbital foramen thus supplying the lower lid region

The sympathetic nerves center in the ciliary ganglion which is placed just lateral to the ophthalmic artery near the apex of the orbit It gives off the short ciliary nerves (p 3619) The pupillary constrictor fibers arising from the third nerve run in these nerves but the dilator fibers by pass the ganglion running from the nasociliary nerve in the long ciliary nerves

EXAMINATION OF THE EYE

Examination of the eye by daylight is supplemented in the darkened room by the use of focal or oblique illumination and ophthalmoscopy

FOCAL OR OBLIQUE ILLUMINATION

The rays of light from an electric bulb 18 inches distant from the patient are concentrated or focussed on the eyeball by a lens of +13 to

+ 18 diopters with a focus of 2 to 3 inches. The lens is grasped by its margin between thumb and index finger and held at the proper focal distance from the eye at such an angle that its surfaces are perpendicular to the source of light. A magnifier is held in the other hand to facilitate examination or a binocular magnifier can be worn like a pair of glasses.

By varying the distance between the lens and the eye the portion to be examined is illuminated clearly. By daylight or oblique illumination the normal structures of the adnexa and outer tunic are noted. Abnormalities are observed and investigated.

NOTES ON THE PHYSICAL EXAMINATION OF THE EYE

1 In the presence of *blepharospasm* one or two drops of 1 or 2 per cent butyn or holocaine are instilled into the conjunctival sac. When anesthesia has been established the conjunctivae may be examined particularly for the presence of foreign bodies.

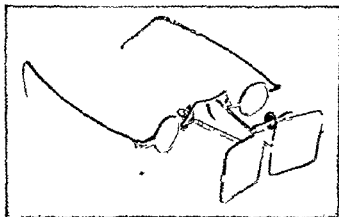


Fig 10.3—Binocular magnifier

2 With the suspicion of disturbances particularly inflammation of the *lacrimal gland or duct* the structures are gently squeezed and resultant discharge is examined bacteriologically by spread and culture.

3 *Patency of the nasolacrimal duct* may be tested by instilling into the conjunctival sac one or two drops of 1 per cent fluorescein. If the duct is normally patent the dye will appear in the nose in a few moments. The conjunctival sac is then irrigated with saline or boric acid solution. A greenish discoloration persists only when the cornea is denuded as the result of laceration or ulceration. The use of the dye is of considerable value in the examination of the cornea and the duct system.

4 The *pupillary reaction to light* is tested with a flashlight. A strong light directed into the pupil produces a miosis which gives way to a mydriasis when the intensity of light is decreased. The *direct reflex* is tested by observing the pupil into which a source of light is directed. The

musculature The orbital muscle of Mueller bridges the inferior orbital fissure and joins the inferior oblique anteriorly Posteriorly it runs under the annulus of Zinn The peribulbar musculature is an indefinite group of smooth muscle fibers surrounding the anterior portion of the globe It is closely connected with Tenon's capsule Anteriorly it extends to the lids

Both these muscles are nonstriated and innervated by the sympathetic Theories concerning exophthalmus have involved their overaction

The Blood Vessels of the Orbit—The blood vessels of the orbit with one exception arise from the ophthalmic artery a branch of the internal carotid They include the central retinal artery the posterior ciliary arteries the supra-orbital the palpebral and ethmoidal branches The infra-orbital artery a branch of the internal maxillary supplying minor branches to the lower contents of the orbit is the exception

The superior ophthalmic vein the largest vein in the orbit empties into the cavernous sinus The inferior ophthalmic vein also does so but a branch goes downwards to the pterygoid plexus Other veins go anteriorly to empty into the facial system

The Nerves of the Orbit—The nerves of the orbit are motor sensory and sympathetic The motor elements are the third fourth and sixth nerves to the extra-ocular muscles and the levator Sensation travels along the ophthalmic or first division of the fifth nerve which enters the orbit through the superior orbital fissure as lacrimal frontal and nasociliary divisions These supply the eyeball lacrimal gland conjunctiva upper lid and surrounding skin



Fig 10b^a—Technic of focal illumination *

The maxillary or second division of the fifth nerve enters as the infra-orbital nerve through the inferior orbital fissure and emerges on the face through the infra-orbital foramen thus supplying the lower lid region

The sympathetic nerves center in the ciliary ganglion which is placed just lateral to the ophthalmic artery near the apex of the orbit It gives off the short ciliary nerves (p 3619) The pupillary constrictor fibers arising from the third nerve run in these nerves but the dilator fibers by pass the ganglion running from the nasociliary nerve in the long ciliary nerves

EXAMINATION OF THE EYE

Examination of the eye by daylight is supplemented in the darkened room by the use of *focal or oblique illumination* and *ophthalmoscopy*

FOCAL OR OBLIQUE ILLUMINATION

The rays of light from an electric bulb 18 inches distant from the patient are concentrated or focussed on the eyeball by a lens of +13 to

9 In diseases of the eye the *preauricular lymph nodes* should be palpated. Enlargement and tenderness bespeak an abnormality in the lids.

10 *Foreign bodies* frequently lodge in the conjunctival sac. They must be sought whenever there is evidence of conjunctival irritation. The membrane of the lower lid is best observed when the patient looks upward while the lower lid is retracted by the examining finger. The upper lid is examined by doubling the structure over the applicator while the patient looks downward. With one hand the physician pulls the upper lashes downward and forward. With the other hand he places an applicator across the upper lid. Simultaneously the applicator is pressed downward and somewhat forward while the lashes are pulled upward and somewhat backward. Successfully executed this maneuver furnishes a satisfactory survey of the conjunctival lining of the upper lid. If the eye is too irritable local anesthesia may be produced by the instillation of 1 per cent or 2 per cent butyn or holocaine.

EXAMINATIONS OF VISION

Visual Acuity—Determination of visual acuity is the easiest and most practical way of estimating the sensitivity and efficiency of the eye. The physician who examines and treats an eye without first determining visual acuity exposes himself to malpractice action. This is particularly important if the treatment is for a compensable injury or involves any legal question.

The determination of the visual acuity is best done by using the Snellen chart. The chart should be well illuminated and the patient placed 20 feet from it. The visual acuity of the right eye is tested first. A card is held by the examiner in front of the left eye and the patient is instructed to read the smallest lines visible to the right eye. The visual acuity is expressed as a fraction whose numerator is the distance from the chart and whose denominator is the smallest line read. Thus if the 20 line is read the vision here would be 20/20. If the best vision is the 50 line it would be 20/50. If two additional letters only are read on the 40 line the vision would be 20/50 plus 2. If the room is not long enough the test should be made at 10 or 15 feet and the vision recorded as 10/15 or 15/20 as the case may be.

If the patient cannot see even the top letter on the chart he should be made to walk up until he sees it and the vision recorded as 4/200 for example. If he sees even less than that he may be tested by counting fingers at one foot distance. If this is not possible testing for hand movements should be done. Failing in this he should be asked to try to recognize light from an ophthalmoscope bulb and the vision recorded as V = P L (light perception) or no P L.

Unless an eye is blind a record of some degree of visual acuity should be made. Both eyes should be tested at all times. If a patient wears glasses he should be tested with and without them.

If the vision is below normal (20/20) the defect may be due to a refractive error or some pathological cause. The easiest way to differentiate between the two is to use the pin hole test. The patient looks through a very small hole in an opaque disk or card. He holds it himself to be sure he gets the hole directly in the center of the pupil. The illumination must

consensual reflex is observed by noting the reaction of the pupil in the opposite eye not exposed to the brighter illumination

5 The reaction to accommodation or more properly the *near reflex* is tested by having the patient look at a far object at which time mydriasis occurs and then focussing on a near object such as the finger of the examiner under which circumstance miosis occurs

The *Argyll Robertson pupil* reacts to the near reflex and not to light



Fig. 1004—Eversion of upper eyelid A First procedure B Second procedure



Fig. 1005—Exposure of upper retro-tarsal fold

Fig. 1006—Eversion of lower eyelid and exposure of lower retro-tarsal fold

6 *Hippus* is alternating miosis and mydriasis of the pupil without any apparent cause

7 *Intra ocular tension* is measured with the *tonometer* (p 1545) The practitioner estimates it by palpation of the globe while the patient looks down with eyes closed This crude test may serve to prevent the precipitation of an acute attack of glaucoma by the inadvertent instillation of a mydriatic (atropine) for diagnostic purposes

8 *Nystagmus* consists of oscillatory movements of the eyes particularly brought out when the eyeball is directed in an extreme position

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acuity This is a special procedure requiring special knowledge and equipment and need not be considered here

Tests for Near Vision and Accommodation—The exact testing of the focussing power or accommodation requires first that the patient wear his distance correction Very small print or a fine single line 0° mm thick and 3 mm long is brought near to the eye and the point at which it becomes blurred is recorded by measurements with a ruler

Color Sense—Testing color vision is of value in congenital color blindness and in examinations for military naval and civil service work especially in transportation The practitioner can usually discover color blindness by having the examinee pick out and match various colored skeins of wool For scientific accuracy testing with a spectral color matching apparatus and pseudoisochromatic charts is necessary

Light Adaptation—Testing the light sense or dark adaptation is a very difficult problem Most of the available instruments have some important defects Apparently the best instrument is the Hecht adaptometer used by research workers The main clinical application is to discover deficiency of vitamin A (p 617) present in the retinal rods The practitioner can often surmise such a defect by asking questions concerning the length of time it takes a patient to be able to find his way about upon entering a moving picture theatre If it is definitely prolonged and no pathological process such as *retinitis pigmentosa* (p 1592) or *glaucoma* (p 1578) is present one may assume a vitamin deficiency

Visual Fields—The investigation of the fields of vision or more properly the limits of indirect vision is a diagnostic procedure of great value Indirect or peripheral vision is vision with those parts of the retina which do not belong to the fovea centralis By examining the visual fields the function of almost the entire retina and its cerebral pathways and end station are studied While the procedure is capable of great refinement much more information can be obtained by a painstaking examiner with crude apparatus than by a haphazard study with an impressive instrument The patient's cooperation is necessary as this is essentially a subjective test

Technic of Examining the Visual Fields *THE PERIPHERAL FIELD*—The confrontation test carefully done provides a considerable amount of information The examiner seated opposite the patient compares his own field with that of the patient One eye of the patient is occluded and the observer shuts his own opposite eye Each fixes his gaze on the open eye of the other The examiner then introduces his hand or preferably a small white object mounted on a handle into the common field of vision in a plane midway between the two Both patient and physician should see the object simultaneously The object is brought in from the periphery until it is seen in a number of meridians around the eye Gross defects in the peripheral visual field are easily discovered and the patient is referred to the ophthalmologist for exact perimetry (p 1541)

See *Differential Diagnosis of Disturbances of Eyelashes and Eyelids* (p 1612) *Conjunctivitis* (p 1618) *Epiphora* (p 1595) *Photophobia* (p 1574) *Headache* (p 1512) *Diplopia* (p 1520) *Abnormalities in Pupillary Diameter* (p 1533) *Pupillary Abnormalities Other than Mydriasis and Miosis* (p 1534) *Diplopia* (p 1520) *Ptoxis* (p 1649) *Disturbances*

of Color, Light and Form Sense (p 1535) Exophthalmos (p 1575) Papilledema (p 1579) Pain in the Eye (p 1582) Disturbances of the Orbit (p 1615) Reduction in the Acuity of Vision (p 1638) Disturbances in the Fields of Vision (p 1645) Vertigo (p 2020)

Ophthalmoscopy—Ophthalmoscopy is easily learned and is of such great value to the practitioner that he should include its use in his routine examination. Ophthalmoscopes while admittedly expensive last a lifetime and furnish invaluable information relative to the eye and the vascular and nervous systems. The retinal blood vessels and the optic nerve alone of the vascular and nerve structures can be visualized in this manner. The earliest lesion of arteriosclerosis is seen in the fundus. Retinal changes

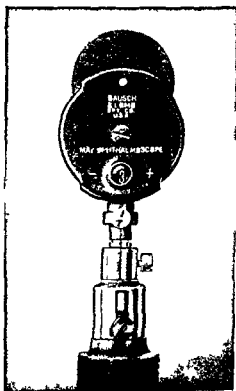


Fig 1050—The May ophthalmoscope *

are observed early in hypertension, nephritis, brain tumor, and syphilis. They are often of great diagnostic and prognostic assistance in tuberculous meningitis, malignant hypertension, the nephropathies, subacute bacterial endocarditis, and sinusitis.

The ophthalmoscope consists of an illuminating system designed to throw light into the interior of the eye and an aperture usually situated above the source of light which allows some of the light returning from the patient's eye to enter the eye of the examiner. The direct ophthalmoscope in general use is the self-luminous variety equipped with an electric bulb and the totally reflecting May prism.

Indirect ophthalmoscopy is difficult to perform and its use is limited to those specialists who use it as an *additional means* of studying the fundus

In the darkened room quite a satisfactory examination of the undilated pupil may be obtained. The right eye of the observer is used to examine the right eye of the patient and the left for the patient's left eye. If a clear view cannot be obtained a mydriatic such as 2 per cent homatropine hydrobromide is instilled into the conjunctival sac. After examination 0.5 per cent eserine (physostigmine salicylate) is instilled to prevent possible elevation of intraocular tension.

Examination of the Media—To examine the ocular media the zero lens of the ophthalmoscope is used at a distance of 12 to 15 inches from the patient. In the normal eye a homogeneous orange red fundus reflex is obtained. Opacities in the media which obliterate part of the light are apparent as dark or black spots upon the colored background. Movable or floating opacities are situated in the vitreous. Fixed opacities which remain stationary when the examiner's head is moved from side to side are in the plane of iris. Those in front of the iris appear to move in the direction opposite to the observer's movement while those in back of the iris move in the same direction.

Examination of the Fundus—To examine the fundus the patient is instructed to look at a distant object over the shoulder of the examiner. The examiner first places the ophthalmoscope about 1 inch from the eye and inserts a +12 diopter or +15 diopter lens in the aperture. In the eyes of most patients the ophthalmoscope is thus focused for a magnified (14x) study of the crystalline lens and anterior vitreous. As the examiner gradually weakens the strength of the lens the posterior vitreous is examined and finally the retina is brought into focus. This procedure permits the recognition of fine opacities in the lens and vitreous. Often this is the only way a vitreous haze in low grade uveitis (p. 1637) is brought to light. Structures such as persistent hyaloid remnants and proliferating vessels are seen clearly.

Many ophthalmoscopic errors of omission as well as misinterpretations of elevation or depression of retinal lesions are based upon the usual procedure of beginning the ophthalmoscopic study of the fundus with a zero lens in the aperture. Occasionally in high astigmatism or myopia a clearer picture is obtained when the patient wears his glasses.

1 *The Normal Fundus*—The beautiful picture that the human fundus presents is made up of a stippled orange red surface upon which the optic disk, the blood vessels and the macula make a complex floral design. The normal fundus varies within great limits.

THE OPTIC DISK (PAPILLA)—The disk or nerve head is considered as to size, shape and color as well as the character of the margins and the physiological cup.

SIZE AND SHAPE—The disk is usually circular with a diameter measuring about 1.5 cm. Sometimes it is congenitally oval generally associated with astigmatism.

COLOR—The disk is usually light grayish or yellowish red in color.

MARGINS—The margins of the optic disk are usually sharply defined.

especially temporally. Nasally more nerve fibers pass over the margin and thus obscure it. For the same reason the inner half of the nerve head looks redder than the outer half. Circumscribing the disk two concentric rings distinct in color are often seen. Lying next to the disk margin is the white *scleral ring* formed by exposure of the sclera when the opening for the optic nerve in the choroid is larger than that of the sclera. The outer or choroidal ring is black and usually incomplete. The dark color is the result of the accumulation of pigment at the border of the choroidal opening for the nerve.

If the optic nerve is displaced to the side in the sclerochoroidal canal or if the canal runs obliquely the *scleral crescent or conus* is seen. A *choroidal crescent or conus* usually on the temporal side of the disk is not pure white but light colored and is due to absence of pigment epithelium in front of it. Both crescents occur mainly in myopia. An *inferior white crescent* is sometimes observed at the lower border of the disk. This is congenital and usually associated with slight impairment of vision even with proper glasses.

Lesions of the disk occur with primary and secondary optic neuritis with increased intracranial tension and increased intraocular tension.

THE PHYSIOLOGICAL CUP—In the center of the optic disk is a funnel shaped depression the physiological cup produced by the separation of the nerve fibers. This is whiter in appearance than the rest of the disk since at the bottom the white lamina cribrosa appears. Blood vessels ascend on the inner wall of the funnel. The cup or excavation may be very large and deep. Sometimes it involves practically the entire outer half of the disk. The excavation never extends entirely to the margin of the disk. In contrast to the pressure excavation of glaucoma or optic atrophy a small marginal rim of nerve is always present.

THE RETINAL BLOOD VESSELS—The central retinal artery and vein usually divide into *superior and inferior divisions* on the surface of the optic disk and each of these soon divides into *nasal and temporal branches*. These pass into the retina where they keep on branching in an arborescent fashion into terminal twigs which do not anastomose. The arteries differ from the veins in that they are bright red in color, smaller in caliber and run a straighter course. They have a bright reflex or light streak running along the center.

Abnormalities of the arteries include *arteriosclerosis, occlusion, angiospasm, narrowing and tortuosity and pulsation*.

The veins are darker of greater caliber (ratio 3:2) and more tortuous. Venous pulsations on the disk may be transmitted to the main arterial stem but otherwise arterial pulsations are abnormal.

Venous abnormalities include *dilatation and engorgement and obliteration* at the arterial crossings with increased venous pressure of local or systemic distribution.

THE RETINA—The retina is transparent in the normal state. In children numerous vivid dancing reflexes often exist especially along the vessels giving the retina the luster of watered silk. In old people the retina is less transparent and less lustrous.

The color of the retina depends on the structures lying behind it, namely the pigment epithelium, the choroid and the sclera.

If the pigment epithelium is profuse the fundus may appear brownish red or brownish gray as in Negroes. If less pigment is contained in the epithelium the accumulations of pigment in the intervacular spaces of the choroid appear as dark islands separated by red bands the choroidal vessels. This is the *tessellated fundus*. In the blond fundus due to absence of pigment the vascular network of the choroid is seen distinctly and the white clera shows through in the interstices of the network.

The Macula Lutea—Very few distinctive features are present at the macula lutea. It is situated a little less than two disk-diameters temporal to the optic nerve on a level with the inferior half of the disk. The region is devoid of blood vessels and appears somewhat darker than the rest of the fundus. In its center is a bright point the fovea centralis. Often the macula is demarcated by a bright halo surrounding a horizontally oval dark area the size of the disk.

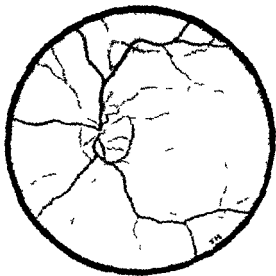


Fig. 1000.—Normal fundus with physiologic cupping.

2 *The Determination of Level in the Fundus*—At times it is necessary to measure differences of levels in the fundus. One valuable method is to note the lens that permits a view of the apex of an elevation for example the edge of a swollen choked disk in papilledema and then note the first lens that allows a clear view of the adjacent fundus. Thus if the disk were seen with a +4 diopter lens and the retina with a -2 diopter lens the elevation would be 6 diopters or 2 mm (3 diopters = 1 mm).

This method will prove fallacious unless study of the fundus begins with a +12 diopter lens as described above. Otherwise the errors introduced by accommodation will be great.

3 *Normal Variations in the Fundus*—Many minor variations occur which only experience will indicate. Medullated nerve fibers constitute a

frequently encountered phenomenon Due to retention of the medullary sheaths of the nerve fibers a whitish striated smudge resembling the hairs of a shaving brush with the optic disk as a handle is seen extending for varying distances toward the periphery Sometimes the medullated area has no connection with the disk

In the congenital condition of pseudoretinitis the disk is redder than usual Its margins are indistinct with slight bulging forward due to marked hyperopia astigmatism or amblyopia Sometimes tortuous veins are present The condition is differentiated from neuritis or papilledema in that the arteries and veins are normal in caliber and the retina is normal

4 *Transillumination of Eyeball*—When an intraocular tumor is suspected transillumination of the eyeball may be very helpful This is simply done The exposed electric bulb of the ophthalmoscope is applied to the external surface of the eyelids with firm pressure against the eyeball This causes the light to shine through the lid and sclera and produces a *reddish glow* in the pupil The light is applied in various directions Any interference with the passage of the light such as by a solid mass will diminish the brilliance of the red glow

INTEGRATIONS

| | |
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| Eye Disturbances of (p 1560) | Epiphora (p 1525) |
| Vision Disturbances of (p 1541) | Ophthalmoscopy (p 1545) |
| Lids Abnormalities of (p 1612) | Pupils Reactions of (p 1534) |
| Conjunctiva Abnormalities of (p 1560) | Intra ocular Tension Abnormalities of (p 1526) |
| Cornea and Sclera Abnormalities of (p 1571) | Strabismus (p 1530) |
| Lens Abnormalities of (p 1592) | Color Blindness (p 1535) |
| Uveal Tract Abnormalities of (p 1632) | Iscrimetry (p 1541) |
| Optic Fundus Abnormalities Observed in (p 1545) | Visual Fields Abnormalities of (p 1645) |
| Retina and Optic Nerve Abnormalities of (p 1640) | Exophthalmos (Proptosis) (p 1575) |
| Orbit Abnormalities of (p 1615) | Enophthalmos (p 1577) |
| | Papilledema (p 1579) |
| | The Ophthalmologist (p 1540) |

TRANSILLUMINATION OF THE BREAST

The female breast may be transilluminated in the dark room The examination is facilitated by a special rubber end piece in the shape of a cone This fits over the point of illumination and prevents burning

The beam of light is placed behind the breast The palpating hand then presses the mammary tissue over the end piece so that each square centimeter of the tissue may be completely explored (*Palpatory transillumination*) It is possible in this way to demonstrate minute lesions The venous channels are also clearly delineated At times they may be traced to a neoplasm

See *Differential Diagnosis of Clinical Disturbances of Breasts and Nipples* (p 2578)

TRANSILLUMINATION OF THE SCROTUM

The scrotum and its contents can be transilluminated using the same technic as for the breast It is possible in this way to detect a solid mass

tumor of the testicle (p 2442) or a *hydrocele* (p 2430) from a *hernial sac* containing bowel (p 3091)

TRANSILLUMINATION OF SUBCUTANEOUS TISSUES

Foreign bodies such as splinters in the finger will cast a shadow by palpatory transillumination

CHAPTER 163

THE PHYSICAL EXAMINATION PELVIS, RECTUM AND REPRODUCTIVE ORGANS

FOLLOWING dark room examination the pelvis rectum and genitalia are investigated by visual and digital examinations Male and female structures require different methods of exploration

THE MALE PERINEUM AND GENITALIA

ANATOMIC REVIEW

The Penis—The penis is composed of three cylindrical masses of erectile tissue bound together by fibrous tissue and covered with skin The two lateral masses are the corpora cavernosa the third or median mass is the corpus spongiosum The corpora cavernosa form the greatest part of the body of the penis In the anterior three fourths they are intimately connected along the median line At the posterior portion they separate to form the *crura penis* two strong tapering fibrous processes or roots that are firmly connected to the rami of the os pubis and ischium A median groove on the upper surface of the corpora lodges the dorsal arteries nerves and veins The groove on the under surface receives the corpus spongiosum The *corpus spongiosum* which contains the urethra is expanded behind to form the *urethral bulb* This lies in apposition to the superficial layer of the triangular ligament

The anterior extremity of the corpus spongiosum is expanded to form the *glans penis* which is molded over the rounded ends of the corpora cavernosa extending further on their upper than their lower surfaces At the summit of the glans is the urethral orifice or *meatus* The circumference of the base of the glans forms the *corona* as a rounded projecting border which overhangs a deep socket behind which is the neck of the penis The *prepuce* is a layer of redundant skin that encircles the neck of the penis and may be drawn over the glans

The penis is divided for descriptive purposes into a root a body and an extremity The root consists of the diverging crura and the mesial bulb of the corpus spongiosum Each corpus is covered by the erector penis The bulb is surrounded by the accelerator urinae The body of the penis extends from the root to the end of the corpora cavernosa while the extremity is formed by the glans covered by the prepuce

The Male Urethra—The male urethra extends from the neck of the bladder to the meatus at the end of the penis Its functions and form are modified by abnormalities in the seminal organs particularly lesions affecting the prostate gland Conversely the reproductive structures are secondarily involved with urinary lesions particularly urethritis of venereal origin The male urethra is subdivided by the external sphincter into an anterior and a posterior portion

Anterior Urethra—The anterior urethra begins at the urethral meatus a vertical slit at the end of the glans penis Normally the meatus is at the summit of the glans Due to congenital abnormalities however it may occupy atypical locations See *Epiispadias* (p 2286) *Hypospadias* (p 2286) The caliber of the urethral meatus is small and the tissue is relatively inelastic It ordinarily approximates 8 mm in diameter It may require splitting (meatotomy) in order to pass instruments larger than 24 F

The first portion of the anterior urethra extends from the meatus to the level of the corona The lumen is fusiform and forms the *fossa navicularis* The neck of this fossa like the meatus may be of small caliber and often requires splitting for instrumentation Folds small valves and reduplications of the mucous membrane which occur in the region of the fossa may produce obstruction to urinary flow They also serve as foci for chronic infection particularly *gonorrhea* The second portion of the anterior urethra is the pendulous division which traverses the corpus spongiosum The caliber of the spongy urethra is fairly uniform throughout The pendulous portion of the anterior urethra gives in to the third or bulbous segment which broadens throughout due to a ventral sagging producing a pouch or bulb At the junction of the bulbous segment with the posterior urethra there wing and

a sharp angulation. These factors produce difficulty in the passage of instruments and favor the formation of inflammatory strictures. Accessory glands are present in the spongy and bulbous segments of the anterior urethra. The submucous glands of Littre open directly onto the surface of the spongy portion. The bulbourethral glands of Cowper open into the bulbous portion. These glands often harbor chronic inflammation particularly in gonorrheal infection.

Posterior Urethra—The posterior urethra leads from the bulbous portion of the anterior urethra to the urinary bladder. It is subdivided into a membranous or muscular portion and a prostatic segment. The membranous portion of the posterior urethra is short. Its junction anteriorly with the bulbous division of the anterior segment is a common site for deep urethral strictures. The external sphincter encircles this part. The prostatic portion of the posterior urethra extends from the vesical orifice posteriorly to the membranous urethra anteriorly. This segment is surrounded throughout by the prostate gland. The lumen of the prostatic urethra is dilated. Many ducts of the seminal organs open directly into it.

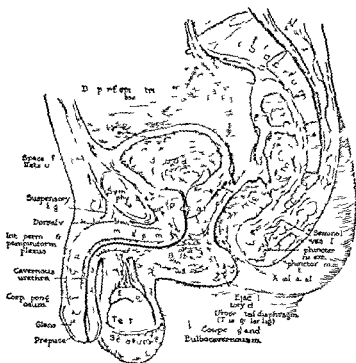


Fig 1061—Medial sagittal section of the male pelvis viewed from the left. The bladder is somewhat distended.

The floor of the prostatic urethra is dilated into a crest, the *verumontanum*. To the right and left of this structure the prostatic sinuses are recognized. Into these the majority of the prostatic ducts open. The verumontanum or urethral crest is itself produced by the ejaculatory ducts and the ureter.

The Urinary Bladder—The urinary bladder functions as a reservoir. It is designed to favor urinary continence. The position of the adult bladder is mostly pelvic. The fundus rises above the level of the symphysis pubis only when it is distended. The lumen of the bladder is Y-shaped when empty or star-shaped when full. The vesical neck contains the orifice connecting the bladder to the posterior urethra. It possesses an internal sphincter.

Within the bladder the vesical neck forms the apex of the trigon. This triangular area has as its base the interureteric ridge (Mercier's base). The ureteral orifices are situated at each lateral margin of the interureteric ridge. Each ureter runs an oblique course through the

bladder wall and for a variable distance along the interureteric ridge. There is thus formed a membranous crescentic fold which acts as a valve to prevent urinary backflow. The remainder of the bladder is made up by the base or fundus, the right and left lateral walls, the anterior wall and the vertex or dome.

The bladder is lined by a smooth mucous surface. Beneath is a muscular wall called the *detrusor vesicae*. This is attached to the back of the pubis at the neck of the bladder much like a toy balloon to its wooden mouthpiece.

The bladder receives its main arterial blood from the superior, inferior and middle vesical arteries, branches of the anterior division of the hypogastric. The bladder veins do not accompany the arteries. They empty anteriorly into the plexus of Santorini and the lateral plexuses.

The innervation of the bladder is of great complexity. The *puddendal nerve* sends motor branches to the striated muscle. *Efferent nerves* are supplied through adrenergic (p. 1394) and cholinergic (p. 1394) divisions of the involuntary nervous system (p. 1388). The former furnishes inhibitory impulses to the detrusor muscle and motor fibers to the trigone, the internal sphincter and the smooth muscle of the proximal part of the urethra. Thus adrenergic drugs such as adrenalin favor retention of urine. The cholinergic system supplies motor fibers to the detrusor muscle and inhibitory fibers to the internal sphincter. Thus the two divisions of the involuntary nervous system are mutually antagonistic in their action. Cholinergic drugs such as physostigmine stimulate the mechanisms of micturition. The depressants such as atropine behave like adrenalin.

The Ureters—The ureters are relatively thick walled muscle tubes with a narrow lumen. They conduct the urine from the pelvis of the kidney to the bladder.

The ureters vary in length, being about 28 to 34 cm. in men and 27 to 29 cm. in women. The right ureter is usually about 1 cm. shorter than the left due to the lower position of the right kidney.

Each ureter has three points of physiological narrowing. These exist at the *ureteropelvic junction*, at the point of crossing of the iliacs and in the *paravesical portion*. Between the first two narrowings is the *abdominal spindle*, between the lower narrowings the *pelvic spindle*. Ureteral stones more readily become impacted at these sites and in the intramural portion.

The Renal Pelvis and Calices—The *renal pelvis* is the upper expanded portion of the ureter. It lines the *sinus renalis*. The pelvis gives into two and occasionally three *major calices*. These subdivide to form four to twelve (average 8) *minor calices*. The minor calices are invaginated by the tip of the *renal papillae* through which the urinary secretion is discharged. The renal pelvis is subdivided into extrarenal and intrarenal portions, the recognition of which is important in the management of *hydronephrosis* (p. 2281).

The capacity of the renal pelvis varies from 5 to 10 cc. An increase of the capacity beyond 15 cc. suggests a pathological dilatation (*hydronephrosis*).

The pelvis and ureters convey urine from kidney to bladder. This is accomplished by a muscular peristalsis beginning at the minor calix and terminating by the forcing of a jet of urine through the ureteral orifice into the bladder. Pyelo-ureteral activity is not merely a matter of simple gravitation. A distinct ureteral rhythm may be recognized by pyeloscopy. Series of systoles and diastoles are recognizable.

As with the bladder, the ureters possess reciprocal autonomic innervation. The *cholinergic stimulants* (physostigmine, etc.) increase ureteral tonus and favor peristalsis. The *adrenergic stimulants* (adrenalin) and *vagal depressants* (atropine) inhibit ureteral peristalsis.

The Scrotum and Tunics of the Testes—The scrotum and the testicular tunics are protective investments of the testes. The scrotum functions as thermoregulator for the testes since these organs undergo atrophy at intra-abdominal temperature.

The skin of the scrotum is thin. It contains sebaceous glands and hair follicles. The surface is divided symmetrically into lateral halves by a *median raphe* which runs from the penis along the scrotum to the anus. *Dermoid cysts* are occasionally encountered in the raphe. From the raphe *rugae* run at right angles corresponding to the dermal muscle fibers. Just beneath the scrotal skin is the *dartos*, consisting of elastic and smooth muscle fibers and connective tissue. The dartos is very vascular and forms a sac for each testis. Careful hemostasis must be observed in injuries or operations involving this tissue. The scrotum varies greatly in size due to the action of the dartos. In elderly and debilitated individuals it tends to be pendulous. Undue warmth has a similar effect. Cold, exercise and emotion cause contraction.

Included as investments for the testes are the *tunicae* which are derived intra-abdominally and transabdominally in the course of the descent of the testes from the intra-abdominal position to its location in the scrotum. The visceral and parietal layers of the *tunica vaginalis*

are derived from peritoneum the infund bulbiform fascia, from the transversalis muscle the cremasteric, from the internal oblique muscle and the intercolumnar from the external oblique muscle. The tunica vaginalis completely invests the testis and epididymis except at the point of contact between the two and posteriorly where the blood vessels and nerves enter. The most dependent part of the scrotum and the tunica are connected by the scrotal ligament.

In the region of testes there are certain vestigial structures mainly of interest to the specialist. These are the appendix testis the appendix epididymis the paradidymus and

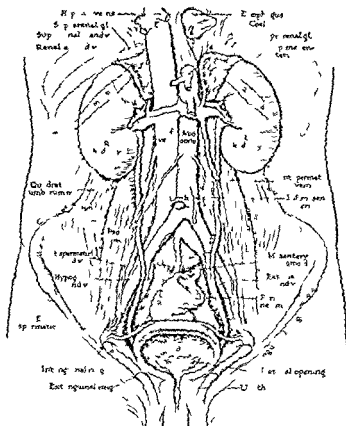


Fig 106 --Topographic relations of urinary system and spermatic cord

the vasa aberrantia. These structures have only embryological significance. Occasionally they become cystic.

The Testis --The testes are almost symmetrical oval grayish-white bodies measuring about 4 cm. in their greatest dimension. They are situated in the scrotum and are surrounded by the testicular tunics. The left testis usually occupies a level lower than that of the right. The long axis of the testis is not quite vertical. It is slightly upward, lateral and forward. The anterior surface is free. The posterior surface is covered by the epididymis.

The structural unit of spermatogenesis is the seminiferous tubule. This structure averages 1 to 3 feet (30 to 91 cm.) though its mean diameter is only 150 to 200 microns. Each testis may have as many as 600 to 1200 seminiferous tubules having a combined length of almost half a mile!

In the cortex of the testis the canals are tortuous and form the ductal convoluts. These

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unite to form connecting canals which in turn form the straight canals or *ductuli recti* and enter the *rete testis*. Within the seminiferous canals the cells show a continuous process of maturation beginning with the *spermatogonium* and proceeding through the *spermatocyte* the *spermatid* and finally the free swimming *spermatozoa*. In this cellular metamorphosis the number of chromosomes of each cell is reduced by one half a process analogous to that which occurs in the ovum during the formation of polar bodies and the ovum.

The entire spermatogenic tubular system is encased by the *tunica albuginea* which sends radiating septa down to the substance of the glands. Within the finer ramifications of the tunica are the *interstitial cells of Leydig* which have an internal secretory function.

The Epididymis.—The epididymides are structures situated bilaterally that cover the posterior surfaces of the testes. Grossly three portions are recognizable the *globus major* or head the *corpus* or body and the *globus minor* or tail. The *globus major* is made up by 12 to 15 efferent ductules which open directly into the ciliated canal of the epididymis. These coils are bound firmly by a framework of connective tissue into a compact body. Eventually the canals open each into the other until beyond the *globus minor* the lumen enlarges to form the *vas deferens*.

Vas Deferens, Spermatic Cord and Vessels of Male Genitalia.—The *vas deferens* or *spermatic duct* a direct continuation of the epididymis is a firm cylindrical muscular tube 15 to 18 inches long. It leads through the spermatic cord and the inguinal canal along the pelvic rim where it winds around the iliac fossa extraperitoneally and loops over the ureter to reach the base of the bladder. Here it enlarges three or fourfold to form the *ampulla* and joins the duct of the seminal vesicles at the base of the prostate to form the *ejaculatory duct*. The *vas deferens* has five recognizable subdivisions: a *testicular portion* contained in the *tunica vaginalis* the *scrotal segment* palpable up to the external inguinal ring; the *inguinal portion* which runs in the canal; the *abdominopelvic division* and the final *vesicoprostatic portion* or *ampulla* which joins with the duct of the seminal vesicle.

The *spermatic cord* is the testicular pedicle extending from the internal abdominal ring through the inguinal canal to the scrotum. The cord has layers corresponding to the tunics of the testis. It contains the *vas deferens*, the blood vessels, nerves and lymphatics of the testes and the epididymides.

The *internal spermatic artery* of the testis arises from the aorta just below the renal artery. Lesser vascular branches are derived from the *deferential artery*, a fine branch of the inferior vesical artery and the cremasteric or external spermatic artery, a branch of the inferior epigastric. The importance of the triple blood supply to the testes rests in the fact that function may be maintained if any one of these vessels is patent though the other two are sacrificed in surgical procedures and in wounds. The *spermatic veins* form the *pampiniform plexus* as a part of the spermatic cord. Dilatation and tortuosity of these veins produces the sensation of a "bag of worms" familiarly recognized as the *varicocele* (p. 2433).

The Seminal Vesicles.—The seminal vesicles secrete a clear gelatinous material that forms part of the seminal fluid. They may also function as reservoirs for sperm. Each sac lies alongside and lateral to its respective *vas*. It has a length of approximately 2 inches and is in close relationship to ureters, bladder and prostate. Each vesicle has a main canal containing many convolutions and diverticula. This canal unites with the *ampulla* of the *vas* to form the *ejaculatory duct*.

The Ejaculatory Ducts.—The ejaculatory ducts are the common ducts formed by the seminal vesicles and the *vas deferens*. They traverse the prostate gland elevating the urethral mucosa to form the *urethral crest* or *verumontanum*. Here, the ejaculatory ducts discharge their contents into the posterior urethra. The prostatic duct opens lateral to the urethral crest.

The Accessory Reproductive Glands.—The mucous structures of Little, the bulbo-urethral glands of Cowper and the prostate gland are accessory reproductive structures.

Little's glands occupy a position in the upper dorsal and lateral surfaces of the urethra. Bacteria may be retained in their acini and function as foci of reinfection. The bulbo-urethral glands of Cowper lie between the prostate and the bulb. These glands form a common duct which penetrates the triangular ligament to open onto the ventral surface of the bulbous urethra. They secrete a mucus rich in albumin which is discharged into the urethra at the time of ejaculation. The main clinical importance of the Cowper glands is their tendency to harbor infection particularly *gonorrhea*.

The prostate gland lies at the neck of the bladder. It is transpierced by the urethra and the ejaculatory ducts. The prostatic gland is five-lobed. The two lateral lobes make up the bulk of the gland. The median anterior and posterior lobes are relatively small. All are

enclosed in a fibrous capsule and make up a mass that is firm and elastic. The prostate varies considerably in size. The average dimensions are 3.4 cm. in length, 4.4 cm. in width and 2.6 cm. in thickness.

The prostate forms an external secretion which is added to the seminal fluid at the time of ejaculation. Its function remains unknown. Its chief significance is related to the urinary obstruction that accompanies prostate hyperplasia, hypertrophy and neoplasms.

CLINICAL EXAMINATION OF MALE PERINEUM AND GENITALIA

The Perineum—The male perineum is scrutinized with the patient recumbent, the legs in stirrup, as for the gynecological examination. *Dermatoses of the perineum, fissures and fistulas* may be encountered.

Anal Orifice—The patient is requested to strain as at stool. *Prolapse of mucous membrane* is noted, as well as the presence of *hemorrhoids, discharge, fissures or fistulas*.

Rectal Examination—The patient is placed in the knee elbow position. Using a lubricated finger cot, the examining finger is introduced into the rectum by a rotary movement so as to cause a minimum of discomfort. The tip of the coccyx may be palpated posteriorly; anteriorly, the integrity of the perineal body is noted. Roughening, induration or irregularity of the mucous membrane is an indication for a *diagnostic proctoscopy* (p. 1907). *Strictures* (p. 1914) are noted. The strength of the anal sphincter is tested by having the patient contract the muscle voluntarily.

Increasing recognition of the frequency of rectal cancer, as well as the common occurrence of other lesions in this region, requires routine digital exploration. It was once said that the difference between the practitioner and the specialist consultant lay in the fact that the latter did a rectal examination.

The finger outlines the lobes of the prostate. The normal prostate gland is about $1\frac{1}{4}$ inches from the base to apex and $1\frac{1}{2}$ inches broad. Its contour is regular, the surface smooth and the consistency equal to that of a ripe peach.

The gland is massaged and immediately thereafter the meatus of the urethra is scrutinized for discharge (p. 2340). This discharge is spread on a slide to be examined for *pus cells* and *gonococci* (p. 217).

The examining finger reaches up laterally for the *seminal vesicles*, which can be felt only when they are full. Any induration or tenderness is worthy of further investigation.

The *supraprostatic rectal mucosa* is carefully palpated for infiltrations or the suggestion of a ridge of indurated tissue, the so-called Blumer's shelf, suggestive of *metastasis* from a carcinoma (p. 1816).

The *ischio-rectal space* laterally is often the site of *abscess* and *fistula formation* (p. 1913). It should be carefully palpated.

Feces—After rectal examination, the examining finger is removed and inspected for blood and feces. The presence of blood requires investigation for the site of its origin. *Fecal specimens for the tests for occult blood* and microscopic study can be obtained from the examining finger (p. 1907).

Impaction of feces may give a clue to the type of constipation.

The Inguinal and Femoral Canals—The male genitalia and the inguinal canals are best studied with the patient standing erect.

The *external inguinal ring* should not admit the tip of the finger. The

presence of any sac or an impulse on coughing requires further investigation. The femoral canal should be intact. See *Hernia* (p 3091).

The Male Genitals—The male genitals are examined by inspection and palpation. Abnormalities require specialist investigation, mostly by visual instrumental methods (p 2254).

The Penis—The penis is carefully inspected with respect to (1) *size* which is correlated with the patient's age and the development of secondary sex characteristics (p 2412) (2) the amount and degree of *pigmentation* and (3) the presence of skin *eruptions* and *ulcerations* (p 2458). Any eruption, particularly one that is *moist*, demands a darkfield examination (p 45). The under surface of the shaft is palpated along the *urethra* for *nodules* and *thickenings*.

The Prepuce—The prepuce is retracted. The accumulation of *smegma* is cleaned away so that the *corona* may be better observed. The *urethral orifice* should be circular, dry and approximately 1 cm in diameter. *Urethral discharge*, *ulcers* and *abnormalities* of prepuce or urethra are further investigated.

The Scrotum—Attention is next drawn to the scrotum, which normally contains both testes, epididymides, vasa deferentia and spermatic veins. Absence of normal structures, *swellings* and *tumors* or *tenderness* demand investigation. *Edema of the skin* occurs early in the course of disturbances of water balance (p 706). *Varicosities* of the veins commonly seen in males of the linear habitus cause the scrotum to hang low. *Tumors and swellings* may be transilluminated. Those containing fluid, such as *hydrocele* (p 2430), light up well. *Solid tumors* of inflammatory or neoplastic origin appear opaque (p 3632).

The Testes—The testes, lying within the scrotal sac, are olive shaped and approximately 1 inch in length. Normally, when the patient stands, the left testis hangs slightly lower than the right. Neither testis should hang down more than an inch below the tip of the penis.

The testes are equal in size, smooth, soft or elastic in consistency and extremely tender on pressure between the thumb and forefinger. Failure to palpate either testis within the scrotal sac may be due to *atrophy*, *agenesis* or *cryptorchidism*. *Pain*, *tenderness* and *swelling* call for further investigation. Absence of pain on pressure suggests *tuberculosis* (p 1464).

The Epididymis—The region of the epididymis is palpated at the upper pole of each testis. It is soft in consistency, nontender and should be no larger than the tip of the little finger. Any *swelling* or *tenderness* is investigated further.

The Vasa Deferentia and Spermatic Veins—The vas deferens is palpated throughout its external length and traced through the external inguinal ring. It is readily rolled between the examining fingers and should be no thicker than the lead of a large pencil. While the vas is palpated, the *spermatic veins* are also examined for *varicosities* and *redundancy*.

See *Differential Diagnosis of Dermatoses of the Genitals and the Perineum* (p 3274). *Ano-perineal Pain* (p 1913). *Incontinence of Feces* (p 1915). *Pruritus Ani* (p 1916). *Retention of Urine* (p 2264). *Incontinence of Urine and Enuresis* (p 2265). *Pain in the Hypogastrium* (p 2302). *Swellings and Tumors of the Hypogastrium* (p 2621). *Hematuria* (p 2620). *Frequency of Urination* (p 2310). *Dysuria*, *Urgency and Strangury* (p 2325). *Urethral Discharge* (p 2340). *Pyuria* (p 2352). *Impotence* (p 2409). *Male Infertility* (p 2419). *Pain in the Scrotum and Testes* (p 2430). *Swellings and Tu-*

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THE BULBI VESTIBULI—The bulbi vestibuli are two masses of erectile tissue containing a plexus of veins situated behind the labia minora on either side of the vaginal orifice. Their vascular connections anastomose with those of the clitoris.

THE VESTIBULE—The vestibule refers to the space between the labia minora. It is bounded above by the clitoris and below by the fourchet. Within it are encountered the

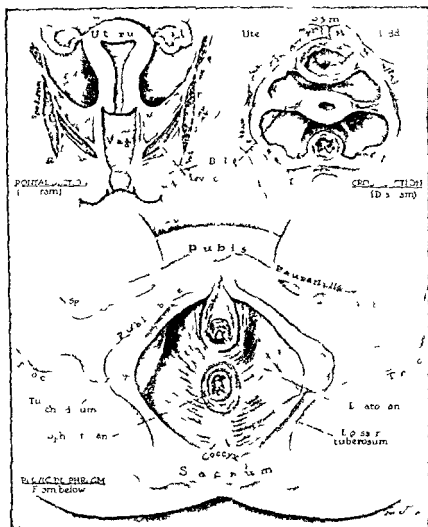


Fig 1064—Relation of pelvic soft parts to skeleton

external meatus the hymen the vaginal orifice and the openings of Skene's and Bartholinian ducts.

THE URETHRA—The female urethra pierces the vestibule about 2 cm. below the clitoris. The urethral canal is approximately 4 cm. in length and extends beneath the pubic arch in a straight or slightly curved direction nearly parallel with the pelvic rim. It is composed of three coats of unstriped muscle and lined by longitudinal folds of squamous epithelium.

Courtesy of S. H. Camp & Company

The *pelvic floor* or diaphragm closes the outlet of the bony pelvis. It forms the musculo-tendinous partition between the pelvic cavity and the perineum. The *pelvic diaphragm* is made up of the levator ani and coccygeal muscles and is pierced by the urethra, vagina and rectum.

Pelvic Measurements—The following may be considered the average measurements of the normal female pelvis:

External

| | |
|-------------------------|-------------|
| Interspinous Diameter | 24 to 26 cm |
| Intercristal Diameter | 27 to 29 cm |
| Bitrochanteric Diameter | 30 to 32 cm |
| External Conjugate | 20 to 21 cm |
| Oblique Diameter | 21 to 22 cm |

Internal

| | |
|----------------------------|-------------------------------|
| Diagonal Conjugate | 12 to 12.5 cm |
| True Conjugate (estimated) | the diagonal less 1.5 to 2 cm |

Outlet

| | |
|--|---------------|
| Anterior Posterior Diameter | 11 to 11.5 cm |
| Transverse Diameter | 10 to 10.5 cm |
| Anterior Sagittal Diameter | 5 to 6 cm |
| Posterior Sagittal Diameter (variable) | 11 cm |

The female pelvis may be classified morphologically particularly as distinguished by radiographs into four main types: (1) the *gynecoid* type which refers to the average form having the accepted female characteristics; (2) the *android* type or masculine figuration; (3) the *anthropoid* type which resembles the transversely contracted pelvis of the great ape; and (4) the *platypelloid* type which refers to the simple flat pelvis.

The female pelvis may be distinguished from the male pelvis by the following characteristics: it is lighter, shallower and lower and less funnel-shaped than the male; the brim is more heart-shaped; the anterior superior iliac spines are more widely separated; the cavity is roomier; the pubic arch is wider; the pelvic inclination is greater; and the ischial tuberosities are further apart.

The Female Perineum—Superficially the perineum is bounded by the mons veneris in front, the buttocks behind and the thighs laterally. It contains all the structures within the pelvic outlet and may be divided into (1) the urogenital triangle and (2) the anal triangle.

The Urogenital Triangle—The urogenital triangle contains the urethra and vaginal orifices and external genitalia. The urogenital diaphragm or triangular ligament is bounded by the ischiopubic rami and bischiatic line and contains the urethral sphincter, the deep transverse perineal muscles and the pudendal vessels and nerves.

The Anal Triangle—The anal triangle is bounded by a line between the ischial tuberosities and the sacrotuberous ligaments and the margins of the gluteus maximus and the coccyx. Its contents include the anal canal, the external sphincter of the anus, the anococcygeal body and the ischiorectal fossae.

The Female Genitalia—*The Vulva*—The vulva is composed of the mons veneris, the labia majora, the labia minora, the clitoris, the bulbi vestibuli, the urethra and Skene's ducts, the hymen and the Bartholinian glands.

MONS VENERIS—The mons veneris is a fatty prominence overlying the symphysis pubis. It is covered with crisp, curly hair arranged in the form of an inverted triangle in contrast to the male escutcheon in which the hair extends upward toward the umbilicus.

LABIA MAJORA—The labia majora are two rounded folds of skin extending from either side of the mons veneris to the posterior commissure or fourchet.

LABIA MINORA—The labia minora or nymphæ are smaller cutaneous folds which embrace the clitoris above and extend downward internal to the labia majora to merge with them in their lower third.

THE CLITORIS—The clitoris, counterpart of the male penis, is a small cylindrical body situated in the median line immediately below the anterior commissure of the vulva. It is composed of two closely approximated crura, capped by an acorn-shaped glans. The crura diverge to lie in close apposition with the lower border of the pubic and ischiatic rami.

THE BULB OF THE VESTIBULE—The bulb of the vestibule are two masses of erectile tissue containing a plexus of veins situated behind the labia minora on either side of the vaginal orifice. Their vascular connections anastomose with those of the clitoris.

THE VESTIBULE—The vestibule refers to the space between the labia minora. It is bounded above by the clitoris and below by the fourchet. Within it are encountered the

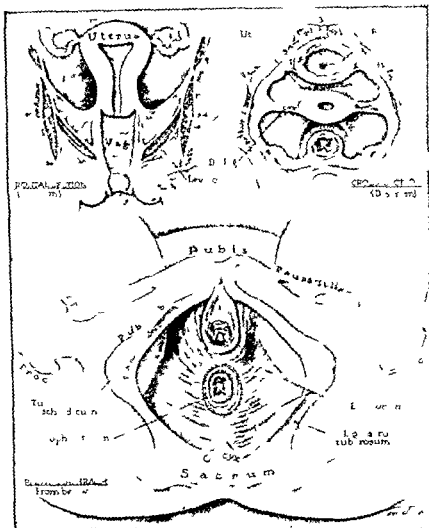


Fig 1084—Relation of pelvic soft parts to skeleton

external os of the uterus, the vaginal orifice and the openings of Skene's and Bartholin's glands.

THE URETHRA—The female urethra pierces the vestibule about 2 cm below the clitoris. The urethral canal is approximately 4 cm in length and extends beneath the pubic arch, in a straight or slightly curved direction nearly parallel with the pelvic rim. It is composed of three coats of unstriped muscle and lined by longitudinal folds of squamous epithelium.

Courtesy of S. H. Camp & Company

SKENE'S DUCTS—Skene's peri urethral ducts are narrow canals 0.5 to 3 cm in length, situated in the lateral aspect of the urethral floor. Their orifices lie just at or within the *urethral meatus*.

THE HYMEN—The hymen is a perforated fold of mucous membrane separating the vestibule from the vagina.

THE BARTHOLINIAN DUCTS—The Bartholinian vulvovaginal ducts open into the vestibule in the groove between the hymen and the labia minora at about the posterior third of the lateral wall. They extend for a distance of 0.5 to 2 cm backward and outward to the deeply situated Bartholinian gland.

The Internal Genitalia—**THE VAGINA**—The vagina is a muscular membranous tube lined by squamous epithelium which connects the uterus and the vulva. At rest, the anterior and posterior walls lie in apposition. The vagina is curved with its convexity directed back.

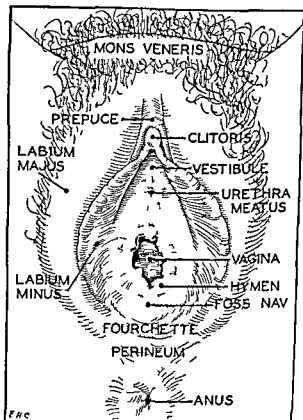


Fig 1065—External genitalia of a normal nulliparous woman

ward. The anterior wall measuring 3 inches is approximately one half shorter than the posterior wall. The expanded upper portion of the vagina or vault which forms an annular groove around the cervix is divided into an *anterior posterior* and two *lateral fornices*. The roof of the posterior fornix contacts the floor of the posterior cul-de-sac of Douglas.

THE UTERUS—The uterus is a pear shaped hollow muscular structure inserted into the vault of the vagina at nearly right angles to the axis. It is roughly 3 inches in length 2 inches in width and 1 inch in thickness. It normally occupies an anteverted and anteverted position and is freely movable. The uterus may be divided into a body isthmus and cervix. The *corpus uteri* is composed of a thick wall of interlacing unstriped muscle covered by a thin glistening peritoneum and lined by a smooth moist mucous membrane. The endometrium is made up of a cellular stroma covered by a single layer of columnar epithelium, from which tubular glands extend down toward the basalis. The *isthmus* of the uterus is the intermediate

zone between the corpus and cervix just at or below the internal os. The cervix is mainly fibrous and represents the passive portion of the uterus. It is lined by mucus-secreting columnar epithelium which forms compound tubular racemose glands. The cervix is divided into an *infra* and *supravaginal* portion.

The ligaments which contribute to uterine support include (1) the broad ligaments (2) the infundibulopelvic ligaments (3) the round ligaments (4) the uterosacral ligaments (5) the utero-vesical ligaments and (6) the cardinal ligaments.

THE FALLOPIAN TUBES—The fallopian tubes or *oviducts* are bilateral muscular tubes, which extend outward from each corner of the uterus along the superior margin of the

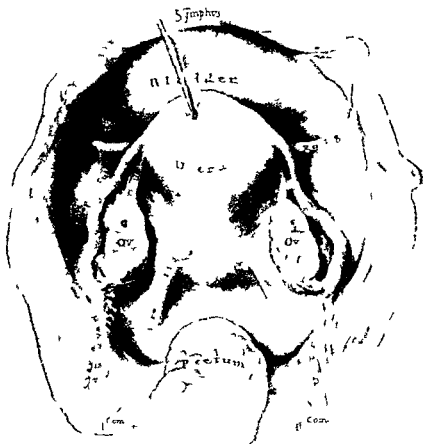


Fig 1066—Pelvic viscera in the female. Viewed from above.

broad ligament for a distance of $3\frac{1}{2}$ to 5 inches. Each tube is divided into an *isthmus* or inner third, the *ampulla* or next succeeding one-half, and finally the *infundibulum* or *fimbriated extremity*. Histologically, the tube is composed of an outer peritoneal lining, two strata of smooth muscle arranged in an inner circular and outer longitudinal layer, and a mucous membrane thrown into multiple longitudinal folds and covered by ciliated, columnar epithelium.

THE OVARIES—The ovaries are bilateral solid organs situated on either side of the uterus, on the posterior aspect of the broad ligament below the level of the fallopian tubes. They

Courtesy of S. H. Camp & Company

are elongated flattened almond shaped and roughly 1 1/4 inches long 3/4 inch wide and 1/2 inch in thickness

The histologic components of the ovary may be said to include (1) a single surface layer of low cuboidal cells which forms the surface epithelium (2) a poorly demarcated connective tissue capsule the *tunica albuginea* (3) a cellular ovarian parenchyma which may be divided into an outer cortex and internal medullary region The cortex contains the *follicular system* in varying stages of growth and degeneration and the medulla includes the blood vessels nerves and lymphatics and such embryonal remnants as the rete ovarii

ARTERIAL SUPPLY OF THE INTERNAL GENITALIA—The uterus and fallopian tubes receive their blood supply from the two uterine and two ovarian arteries The ovaries are nourished mainly by the ovarian division The vagina is supplied by the vaginal branch of the uterine artery the middle hemorrhoidal and the internal pudendal arteries

LYMPHATIC DRAINAGE—The lymphatics of the vulva and lower third of the vagina empty into the inguinal lymph nodes The upper two-thirds of the vagina and cervix are drained by the hypogastric nodes and those nodes about the common and external iliac vessels The uterine lymphatics extend through the broad ligaments to join the ovarian lymphatics to the aortic nodes In addition they drain into the hypogastric common iliac and external iliac nodes

NERVE SUPPLY—The bladder internal genitalia and rectum are innervated by the hypogastric plexus of the sympathetic nerves the sacral ganglia and the gray communicating branches from the second third and fourth sacral nerves The ovaries are supplied by the renal and aortic plexus of sympathetic nerves and the perineum mainly by the pudendal nerve

EXAMINATION OF FEMALE PERINEUM PELVIS AND GENITALIA

Anus Perineum and Rectum—The female anus perineum and rectum are examined exactly as in the male (p 3640) By rectum the cervix and uterus can be readily palpated

The perineum and the ischiorectal regions are closely scrutinized for eruptions evidences of *fissure fistulae prolapse* (p 2542) or *discharge*

Pelvimetry—Pelvimetry (p 2642) is practised in obstetrical cases

Genitals—The bladder and rectum should be emptied just before the pelvic examination Before the pelvic examination the female is requested to stand on the footstool before the examining table The inguinal and femoral regions are observed particularly following cough to detect the presence of *hernia* (p 3091)

In the presence of the aide or nurse the woman then lies on the examining table and places her feet in the stirrups She is properly draped so that the examination of the genitalia and perineum may be accomplished without embarrassment or unnecessary exposure A large pillow under the head assists abdominal relaxation

The Vulva—The vulva is inspected If the patient is virginal and a *hymen* exists digital examination should not be performed unless there is special indication and unless permission is granted by the patient or if the latter is a minor by her mother

Vaginal Discharge—Using a good light the *labia* are separated *Eruptions* and particularly *ulcerations* are carefully noted *Vaginal discharge* is collected and spread on slides for direct and stained examination (p 2496) The patient is instructed to strain so that weakness of the anterior and posterior vaginal walls may be determined

Clitoris—The clitoris is inspected and palpated Abnormalities are noted and if possible corrected (p 2529)

Urethral Meatus and Urethra—The urethral meatus should be visible just below the clitoris Its orifice is examined for *discharge* or *granula*

tions The examining finger is then inserted along the course of the urethra which is milked. The meatus is again inspected for discharge which may then be examined under the microscope (p 2340)

Skene's Ducts and Glands of Bartholin—The region of the Skene's ducts is observed laterally at the orifice of the meatus. The glands may be gently palpated. Discharge resulting from pressure on the glands is collected and examined. Further down in the lateral walls of the vulva may be felt the *glands of Bartholin*. These are palpated. Any discharge, swelling or infiltration is to be investigated.

Vagina—The vaginal speculum is gently inserted and the cavity examined by means of a good light. Large cotton applicators should be readily at hand so that discharge may be completely wiped away. The color of the *vaginal wall* is noted. The source of the vaginal discharge may

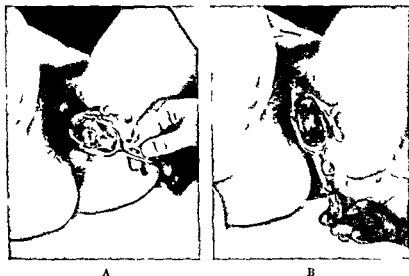


Fig 1067—A Shows introduction of bivalve speculum B The bivalve speculum in place

be ascertained by cleansing the entire vagina and observing the source of the fresh discharge.

Relaxation of the walls is best observed by asking the patient to cough. Bulging of the upper wall suggests a possible *cystocele* (p 2537) of the lower a *rectocele* (p 2537).

Cervix of Uterus—The cervix of the uterus should readily come into view. In the nulliparous the cervical os is a mere dimple. In those who have had surgical procedures such as abortion or who have given birth to children the cervix and the os may be enlarged, patulous, ulcerated or scarred. Any *discharge* from the cervix is carefully examined.

A tendency of the cervical lip or of tissues that surround it to bleed easily suggests neoplasm and warrants consultation with a specialist.

Internal Genitals—The inspection of the external parts of the vagina and uterus having been completed, the index and middle fingers are then

inserted within the vagina and the free hand is used abdominally so that the pelvic viscera are outlined

Technic of Bimanual Examination—In the performance of the abdominovaginal bimanual examination the left fornix is best examined by the physician's right hand vaginally, the left hand abdominally. The positions are reversed for the right fornix. Rectovaginal and rectoabdominal examinations may also be made by the bimanual method. A loop of bowel particularly in the visceroprotic patient may be felt by the examining finger palpating the vaginal fornices and gives the impression of a tumor of the reproductive system. The presence of feces in the rectum gives rise to similar confusion.

The *cervix* is carefully palpated with the vaginal hand for nodules and irregularities. The consistency is elastic resembling that of the tip



Fig. 1068.—The bimanual examination *

of the nose. It is pressed downward and backward so as to raise the body of the uterus to the vicinity of the abdominal hand. When the uterus is in its normal anteverted position it is possible to grasp it between the abdominal and the vaginal hands. The uterus measures about 3 inches in length, 2 inches in breadth, 1 inch in thickness. It should be anteflexed, freely movable and devoid of tenderness or irregularity.

After the body of the uterus has been palpated the supravaginal portion should be squeezed between the two hands and its consistency noted. It is easily compressed in early pregnancy (*Hegar's sign*) and the cervix feels soft (*Goodell's sign*).

The *fallopian tubes* at the point where they join the uterus may be felt between the two hands. They are about 4 inches long and when rolled between the two fingers have the caliber of a pipe cleaner.

The *ovaries* are palpated in each lateral fornix. Each ovary measures $1\frac{1}{2}$ inches in length $\frac{3}{4}$ inch in width and $\frac{1}{3}$ inch in thickness. They are normally slightly tender.

To complete the pelvic examination the genitalia are palpated by rectum by either bimanual rectoabdominal or rectovaginal methods. With retroversion of the uterus this last method is particularly valuable.

See *Differential Diagnosis of Dermatoses of the Genitals and the Perineum* (p. 290) *Anoperineal Pain* (p. 1913) *Incontinence of Feces* (p. 1915) *Pruritus Ani* (p. 1916) *Pruritus Vulvae* (p. 2594) *Retention of Urine* (p. 2264) *Incontinence of Urine and Enuresis* (p. 226) *Pain in the Hypogastrium* (p. 2302) *Swellings and Tumors of the Hypogastrium* (p. 2621) *Hematuria* (p. 2306) *Frequency of Urination* (p. 2310) *Dysuria* *Urgency and Strangury* (p. 2325) *Urethral Discharge* (p. 2340) *Pyuria* (p. 2352) *Inguinal Swellings and Tumors* (p. 3092) *Prepuberal Bleeding* (p. 2479) *Abnormalities of Sexual Development in the Female* (p. 2480) *Disturbances of Female Sexuality* (p. 2491) *Female Infertility* (p. 2492) *Disturbances of the Perineum, Vulva and the Vagina* (p. 2549) *Menorrhagia* (p. 2557) *Dysmenorrhea* (p. 2561) *Metrorrhagia* (p. 2565) *Oligomenorrhea* *Hypomenorrhea* and *Amenorrhea* (p. 2618) *Special Causes of Vaginal Bleeding in Association with Pregnancy* (p. 2664) *Swellings and Tumors on the Right Lower Quadrant* (p. 1886) *Swellings and Tumors in the Left Lower Quadrant* (p. 1870) *Pain in the Left Lower Quadrant* (p. 1866) *Pain in the Right Lower Quadrant* (p. 1880)

CHAPTER 164

THE PHYSICAL EXAMINATION PROCEDURES FOR THE SPECIALIST

SPECIALIST consultation for diagnostic purposes is dependent upon a number of variables. These include (1) the attitude and aptitude of the practitioner (2) the viewpoint of the patient and his friends (3) the skill and equipment of the consultant and (4) the nature of the medical condition.

THE PRACTITIONER AND DIAGNOSTIC CONSULTATION

The *attitude of the practitioner* often determines the extent to which diagnostic consultation is employed. The insecure constantly seek verification of findings whereas the bolder and overconfident regard the request for consultation as a personal affront. The former attitude relegates the practitioner to the role of a *feeder* for his specialist friends while hostility to legitimate consultation may result unhappily for both physician and patient.

Consultation for purposes of diagnosis may be mandatory or optional. *Mandatory* consultation (see p. 3899) is indicated for technical or scientific considerations while *optional* consultation may be held for protection of the practitioner, an academic interest on his part or upon demand of the patient or friends because of their feeling of insecurity.

THE PATIENT AND THE DIAGNOSTIC CONSULTANT

The *attitude of the patient* modifies consultation for diagnostic purposes. Some patients resent being sent to another physician. They believe that the practitioner who is licensed to practice general medicine and to whom they have paid a fee should be in possession of all medical information. They may balk at the suggestion for consultation and seek another practitioner.

Different customs regarding consultation prevail in different communities. In the larger cities patients are *specialist minded*. With or without the knowledge of their general practitioner they may use a different physician for each of their ailments. In urban communities a great deal of the confusion of private practice is due to this faulty method of consultation which deals with each system as a unity and neglects the integration of findings relative to the organism as a whole.

Many awkward situations result from consultation between patient and specialist without the knowledge of the practitioner. The intelligent *sia* diagnose their own difficulties and consult the specialist who to them or their friends seems indicated. This is accomplished without the courtesy or good sense of notifying the practitioner. At times patients distrust conversation between referring doctor and specialist; they fear that the specialist, because of medical ethics, will be forced to agree with his col-

league Many who know of the evil of *fee splitting* believe that the cost of the specialist consultation is greater when the practitioner accompanies the patient

Several courses are open to the practitioner following a consultation from which he has been excluded He may of course never know that such consultation has taken place Usually if things go badly or a surgical procedure is suggested the patient scurries back to the practitioner for consultation regarding the consultant's advice Under these circumstances the practitioner is wiser amicably to re enter the situation and get in touch with the specialist It is better to forget the amenities in the interest of the patient's welfare An opposite course arouses alike the ill will of patient and specialist In smaller communities and in rural districts consultation is rarely sought until the medical resources of the practitioner have become exhausted The specialist consultation may entail hardship sacrifice burdensome expense and a tiresome trip

The patient may be hostile to the *type of consultation* While it is not difficult to persuade the patient to consult an ophthalmologist or otol ogist psychiatric or urologic consultation is often resented The patient or his friends assume that reference to the psychiatrist indicates the belief of the practitioner that the patient is crazy or a faker If he is seen in the office of a psychiatrist he fears that the general public will conclude that he has some mental aberration He likewise may believe that the stigma of venereal disease attaches to his presence in the waiting room of a urologist

QUALIFICATIONS OF THE SPECIALIST

The establishment in 1933 of the *Advisory Board for Medical Specialties* constituted a great step forward in the determination of the competent and able specialists in the various fields of medicine The publication in 1939 of the *Directory of Medical Specialists* makes it simple for the practitioner in any community to be aware of those men who have fulfilled the qualifications for the various boards The Boards include ophthalmology otolaryngology obstetrics and gynecology dermatology and syphilology pediatrics psychiatry and neurology radiology orthopedic surgery urology internal medicine pathology surgery neurosurgery anesthesiology and plastic surgery

The qualifications of the candidates include (1) satisfactory moral and ethical standing in the profession (2) membership in the *American or Canadian Medical Association* (3) graduation from a medical school of the United States or Canada recognized by the Council on Medical Education of the American Medical Association (4) the completion of internship for not less than a year in an approved hospital (5) special training after the internship of not less than three years in clinic dispensary hospital or laboratory recognized by the Council (6) a period of specialized preparation to include graduate training in the basic sciences and active experience in the particular specialty of not less than eighteen months in a recognized institution (7) an additional period of not less than two years of study and/or practice (8) written and oral examination by a board of specialists in the theory and practice of the chosen branch of medicine or surgery

LOCAL VERSUS DISTANT CONSULTATION

The choice of the specialist presents many knotty problems. The practitioner may send his reference work to the local specialist or to the distant out of town specialist or clinic. In either instance he exposes himself to criticism. It is unfair to the patient to insist upon the local specialist if the more competent work can be done elsewhere. However the practice of referring patients out of the community may provoke considerable ill will among colleagues.

Disadvantages of Distant Consultation—Reference to out of town specialists leads to many difficulties for the patient. The expense is greater, transportation is difficult and costly, the cost of treatment if prolonged must be paid by the patient while he is prevented from carrying on part time lucrative work at home. If the therapy requires follow up observations or postoperative dressings and care the patient must remain away for a long time or make frequent trips at great expense. Finally he may take pride in hand and request the local specialist to assume the further care of the medical condition. The latter can hardly be criticized if he does not participate with the greatest good will.

Advantages of Local Consultation—Consultation with local specialists has many advantages. A competent local medical community cannot be built unless the general practitioner loyally supports his neighbor colleague always remembering that the welfare of the individual patient must not be made to suffer. Since emergency surgery is done by the local specialist it would seem only fair to give this colleague the opportunity of dealing with procedures of choice. The community requires the services of a first class surgeon and a first class specialist in the various fields. If these men can be trusted with acute exigencies they should be accorded the courtesy and opportunity of doing the simpler interval procedures.

Local treatment has also the inestimable benefit of personal interest. The patient is near his own home and family, the general practitioner may cooperate in the various problems that arise, the expense of transportation is avoided, a business man is near enough to his interests so that he may conduct his affairs.

CONSULTATION WITH THE INTERNIST AS AN UMPIRE

Differences of opinion often exist between specialist and general practitioner. Under these circumstances the internist may be used as a referee or umpire (p 3901).

The specialist usually has broader indications for operative interference. The internist is apt to be objective concerning operative procedure and like the practitioner tends more to conservatism if he can conscientiously agree with the more conservative attitude of the practitioner. The weight of his opinion assists in the solution of the problem and the responsibility of non intervention is shared.

The practitioner and internist with their more or less similar approach do not always oppose surgical intervention. There are times when the greater risk is surgical conservatism and the safer procedure an operative intervention. Thus with the suspicion that the abdominal condition is a penetration, a perforation or an acute inflammation of a hollow viscus it is safer to perform an exploratory laparotomy than to temporize and risk the

chances of a spreading peritonitis. For the abdominal tumors the more direct procedure is laparotomy rather than prolonged diagnostic investigation. It is wiser to remove one of a group of enlarged lymph nodes and obtain a histological examination than to indulge in diagnostic quibbling.

THE TECHNIC OF CONSULTATION

Ideally the practitioner and his patient agree there is need for specialist consultation. They discuss the choice of the individual specialist preferably choosing one who is a diplomate of the specialty board. The appointment being made the practitioner may accompany his patient to the consultant, give the record orally and observe the examination. If this is not feasible he should prepare a written abstract with the diagnostic data, the progress of events and the results of therapy both helpful and untoward.

Whether the practitioner accompanies the patient to the specialist's office or conveys his information by letter the specialist owes him a written report of findings and recommendations. The practitioner may then decide what therapeutic program to follow and whether to attempt to execute this himself or transfer the patient to the specialist for the completion of therapy.

THE ABUSE OF CONSULTATION

The ideal consultation is the exception rather than the rule. The patient often selects the specialists and as has been said makes his visit without the practitioner who may never learn what has transpired. The patient may insist on choosing the specialist for no other reason than that he is a personal friend or that he cares for a relative. Much more lamentable than this situation is that in which the practitioner employs a specialist for purely personal or selfish reasons. There are no more reprehensible medical practices than those of *log rolling*, interchange of patients and *fee splitting*. The practitioner must be careful particularly in the larger communities to avoid any relationship with the specialist *fee splitter*. The ethical and honest practitioner will certainly refuse to accept any portion of the fee his patient has paid to the specialist. More than that he should not jeopardize his reputation by referring patients to one who is a known *fee splitter* lest the innocent practitioner be charged with the sins of the guilty specialist.

The reference of patients to outside men adds to the practitioner's experience and permits him to broaden his scope. When the practitioner refers patients to the specialist for purely chauvinistic reasons both physician and patient may suffer since a more expert specialist might be one who just does not happen to be a particular acquaintance or friend of the referring doctor.

One of the great difficulties that follows consultation even under the best circumstances arises from the legitimate difference in viewpoint of practitioner and specialist. Many specialists have slit vision. They have little or no interest in any part of the human organism beyond that which is their province. No matter what may be the patient's symptomatology they refer the presenting symptom to findings in their small field without consideration of the other available data. Thus to the dentist all focal

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infection is presumably oral in origin. Many laryngologists advise tonsillectomy for any patient who has skeletal pain no matter what its cause. It is not uncommon for a patient to have a single symptom variously interpreted by several consultants each of whom believes the particular abnormality belongs to his particular field. Arthritics may be told by the dentist that the disease results from an apical abscess by the pharyngologist that it is due to tonsillitis by the orthopedist that it is caused by mechanical difficulties and by the chiropractor, that there is a slipped vertebra.

The practitioner serves great purpose when he integrates specialist findings in terms of his individual patient. This constitutes a happy task when in an obscure or difficult problem the specialist offers a simple and tangible finding with promise of definite relief or cure. It is a thankless chore when the practitioner accepting the positive finding is forced to the conclusion that the abnormality is unrelated to the disturbance or that the suggested procedure has little or no promise of relief. To state to an enthusiastic colleague or a hopeful patient that tonsillectomy will likely not relieve rheumatoid arthritis nor tooth extraction cure sciatica wins few friends and favorably influences fewer people. More often this type of measured and intelligent advice is interpreted by colleague and patient as 'negativism' or defeatism.

MANDATORY DIAGNOSTIC CONSULTATION

The practitioner should insist upon specialist consultation in the following circumstances:

- 1 A *positive finding* in a specialist field. Such might be a tumor of the uterus or a suspicion of optic neuritis.
- 2 A *potentially malignant lesion* such as a nodule of the larynx or in the breast.
- 3 *Failure of the patient to respond to treatment*.
- 4 The production of *untoward symptoms* resulting from attempted therapy.
- 5 The possibility that *complications* may arise later and require the intervention of the specialist as for example impending perforation of a gastric ulcer or a suspected postpneumonic empyema.
- 6 The use of some *apparatus* whose employment demands special skill such as a cystoscope or a bronchoscope.
- 7 Possible *medicolegal* aspects for example commitment of a disturbed or deranged patient.

SPECIFIC INDICATIONS FOR DIAGNOSTIC CONSULTATION

In addition to the mandatory requirements there are specific indications for diagnostic consultation in the various specialist fields. Some examinations require a special technical skill others a wide experience so that data may be correctly evaluated.

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CHAPTER 165

LABORATORY METHODS INTRODUCTION

LABORATORY examinations are an integral part of modern medical practice. Chemists, physicists, physiologists, pathologists, bacteriologists, and physicians have united to simplify technical procedures and to design ingenious apparatus for the assistance of the practitioner in the management of his clinical problems.

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Biopsy (p. 3930)

Pregnancy Tests (p. 2911)

Electrocardiography

- Electrocardiograph (p. 802)
- Electrocardiogram (p. 811)
- Phonocardiograph (p. 806)

Röntgenology

- Fluoroscopy (p. 79)
- Teleoroentgenography (p. 803)
- Simple Radiography (p. 3740)
- Contrast Radiography (p. 3742)

LABORATORY SERVICES

The practitioner may refer his work to the specialist clinical pathologist or obtain assistance from governmental agencies such as the United States Public Health Service or the State and Municipal Boards of Health. In most communities it is possible to obtain serologic tests for

- 4 Over the sink and drainboard a wall fixture of wood provided with *spikes for draining glassware*
- 5 A *table or desk* placed before a window a source of artificial light such as a small electric microscope lamp
- 6 Two sets of *three or four drawers* adjacent to the table or desk
- 7 A *gas outlet* for a Bunsen burner with a length of pressure tubing
- 8 *Several electric outlets* At least one should be placed near the desk for the attachment of the microscope lamp others should be situated in the various corners of the laboratory for the centrifuge and the incubator
- 9 The remaining wall space should have *shelves* for the storage of chemicals and solutions
- 10 A *refuse can* preferably opened by a foot pedal and provided with removable brown paper bags

TABLE 174.—PURE CHEMICALS FOR OFFICE LABORATORY TESTS

| Pure Chemical | Amount (Gm or Cc) | Pure Chemical | Amount (Gm or Cc) |
|-----------------------------------|----------------------|----------------------------------|----------------------|
| Acid Acetic (Glacial) | 500 | Glycerin | 500 |
| Acid Citric (Fahach) | 100 | Iodine (Crystal) | 2 |
| Acid, Hydrochloric (Concentrated) | 500 | Mercuric Chloride | 100 |
| Acid, Nitric (Concentrated) | 500 | Oil of Cedar | 100 |
| Acid Picric (Ebach) | 10 | Phenol | 100 |
| Acid Sulfuric (Concentrated) | 500 | Phenolphthalein | 25 |
| Alcohol, Ethyl | 500 | Potassium Ferrocyanide (Crystal) | 100 |
| Ammonia (23 per cent) | 500 | Potassium Iodide | 100 |
| Benzidine Crystals | 30 | Potassium Sulfate | 50 |
| Calcium Chloride | 500 | Potassium Thiocyanate (Crystal) | 500 |
| Chloroform (Anhydrous) | 500 | Sodium Carbonate (Anhydrous) | 500 |
| Copper Sulfate (Crystal) | 500 | Sodium Carbonate (Crystal) | 500 |
| Dextrose | 500 | Sodium Citrate (Crystal) | 500 |
| Dimethyl amino-azobenzol | 10 | Sodium Hydroxide (Sticks) | 500 |
| Ether | 500 | Sodium Nitroprusside | 100 |
| Ferric Chloride | 100 | Sodium Tungstate | 100 |
| Formaldehyde (37 per cent) | 100 | Xylene | 500 |

- 11 An *electric or gas refrigerator* for the storage of serums and biologicals A corner in the family ice box may be used for the same purpose
- 12 A *floor covering of linoleum* that can be cleaned with a wet mop
- 13 A small *electric incubator* provided with a thermostat
- 14 A small *electric centrifuge* and *centrifuge tubes*
- 15 An *inexpensive torsion balance*

The carpentry plumbing and electric work may be done by local crafts men supplies can be purchased from the local dealer in surgical equipment If there is no available agency they may be obtained by mail order from the larger supply houses

The Storing of Material—The pure chemicals that are not in frequent use may be stored alphabetically on the highest or lowest shelves of the laboratory storage cabinet the solutions for ready use are placed on the

syphilis sputum typing for pneumonia examination of specimens for tubercle bacilli histological reports on excised tissue stool examinations for the bacterial and helminthic invaders serum agglutination reactions and roentgenograms of the chest as part of public health programs

THE OFFICE LABORATORY

The extent to which the individual physician equips his office laboratory varies with his training requirements and personal preferences In some communities laboratory assistance is readily available elsewhere the practitioner is wholly dependent upon his own resources

The physician whose main interest is internal medicine has greater need for his own laboratory than the colleague whose bent is toward obstetrics gynecology or psychiatry Men with established practices have neither the time nor the surplus energy to devote to laboratory work while the tyro in the early years of practice may profitably utilize free hours in the establishment and maintenance of an office laboratory

The deterrents to the establishment of the office laboratory are (1) the capital investment required for the purchase of apparatus and materials (2) the confusion that results from the multiplicity of tests described in the texts (3) lack of guidance in the purchase maintenance and care of equipment and material The presentation that follows aims to overcome these objections and difficulties

The establishment of the office laboratory should be a boon from many diverse standpoints Scientifically many of the laboratory procedures are imperative for good practice as exemplified by the early diagnosis of incipient diabetes mellitus when the urine analysis reveals glycosuria The complete control of the laboratory gives a satisfaction that more than compensates for the difficulties inherent in its establishment Colleagues and lay members of the community appreciate the availability of the laboratory which brings scientific advances to the community and patients have a sense of security in the knowledge that the requisite tests are readily accessible

The Economics of the Laboratory —The capital expense required to establish the office laboratory may seem prohibitive The cost of maintenance is relatively slight however and before long the office laboratory should prove self supporting and revenue producing

The busy practitioner who is well established may take in a junior assistant to set up and maintain his laboratory Older colleagues are often delighted at the opportunity of having specimens examined by a reliable and well trained younger physician The interchange is in the interest of good will and is of invaluable financial aid to the beginner

Housing the Laboratory —The office laboratory requires little floor space The kitchen a bathroom or a spare bedroom may be used in an apartment building or private house

The laboratory should contain

- 1 A wash basin preferably a sink with hot and cold running water
- 2 A water suction pump attached to the tap and furnished with a length of pressure tubing
- 3 Adjacent to the sink, an ordinary kitchen drainboard

TABLE 15—APPARATUS FOR OFFICE LABORATORY (continued)

| Article | Specifications | Number Needed |
|-------------------|--|---------------|
| Microscope | Standard American type with large base exact adjustments and square-covered edges so that a mechanical stage can be used The sub-stage is to have a swing-out condenser the mirror to be adjustable in 2 planes 2 eye pieces 5 x and 10 x and 3 objectives 16 mm 4 mm and 19 mm for oil immersion Abbe condenser 1.0 N.A. magnification from 50 to 200 diameters | 2 |
| Mortar and Pestle | 4 inches | 1 |
| Pipettes | Measuring 1 cc in $\frac{1}{8}$ " | 6 |
| | 2 cc in $\frac{1}{8}$ " | 6 |
| | 5 cc in $\frac{1}{8}$ " | 10 |
| | 10 cc in $\frac{1}{8}$ " | 3 |
| | 25 cc in $\frac{1}{8}$ " | 1 |
| | Measuring Volumetric 2 cc | 12 |
| | 5 cc | 6 |
| | 10 cc | 3 |
| Platinum Loop | | 1 |
| Racks | For test tubes wood | 2 |
| Slides | For microscope | 1 gross |
| Stoppers | Rubber miscellaneous | |
| Test Glasses | Sediment, 60 cc. | 6 |
| Thermometer | Clinical | 1 |
| Tools | Scissors, hammer file wrench can-opener corkscrew jack knife miscellaneous | |
| Tripod | | 1 |
| Tubes | Centrifuge 15 cc ungraded | 6 |
| | Connecting $\frac{1}{4}$ " | 4 |
| | Fermentation (Fig.) | 2 |
| | Sedimentation | 4 |
| | Test, 6 x $\frac{1}{4}$ " | 49 |
| Urinals | Glass | 1 |
| | Male | 1 |
| | Female | 1 |
| Urinometer | | 1 |
| Waste Pail | 4 gallons the control by foot pedal, with paper bags for lining | 1 |

TABLE 16—SOLUTIONS, CHEMICALS AND APPARATUS READY FOR OFFICE LABORATORY

| Article | Container | Amount |
|--|----------------|---------|
| <i>Line Shelf</i> | | |
| Urinometer | | |
| Measuring Cylinder (1000 cc.) | | |
| Litmus Paper | | |
| Acetic Acid (9 per cent) | Dropper bottle | 120 cc |
| Esbach Reagent (p. 3672) | Bottle | 1000 cc |
| Esbach Tube and Rubber Stopper | | |
| Benedict Qualitative Solution (p. 3674) | Bottle | 1000 cc |
| Benedict Quantitative Solution (p. 3675) | Bottle | 1000 cc |

TABLE 175—APPARATUS FOR OFFICE LABORATORY

| Article | Specifications | Number Needed |
|----------------------|--|---------------|
| Balance | | 1 |
| Beakers | Pyrex glass with spout | |
| | 100 cc | 4 |
| | 600 cc | 2 |
| | 1000 cc | 2 |
| Books | Ledgers | 2 |
| Bottles | Glass stoppered narrow mouth | |
| | 120 cc | 18 |
| | 250 cc | 6 |
| | 500 cc | 6 |
| | 1000 cc | 18 |
| | Dropper 30 cc. | 6 |
| | Wash 1000 cc | 1 |
| | Dropper | 8 |
| Brushes | Test tube | 6 |
| Burner | Micro Bunsen | 1 |
| Centrifuge | Double arm with carriers for 4 tubes and rheostat Specify current and voltage | 1 |
| Colorimeter | For phenolsulfonphthalein | 1 |
| | Duboseq | 1 |
| Corks | Miscellaneous | |
| Counting Chamber | Neubauer bright line ruling with pipettes for red and white cells cover glass and velvet lined box | 1 |
| Cylinders | Measuring glass with lip | |
| | 50 cc | 2 |
| | 100 cc | 2 |
| | 1000 cc | 2 |
| Dishes | Porcelain evaporation | |
| | 50 cc | 6 |
| Esbach albuminometer | | 1 |
| Filter Paper | Width—20 cm | 2 |
| | 40 cm | 1 |
| Flasks | Erlenmeyer | |
| | 500 cc | 4 |
| | 125 cc | 6 |
| | 50 cc | 1 |
| | Volumetric | |
| | 1000 cc | 1 |
| | 500 cc | 1 |
| | 250 cc | 1 |
| | 100 cc | 1 |
| Funnels | Glass with 6 stem 100 mm width | 6 |
| Funnel Stand | | 1 |
| Hemoglobinometer | Sahl | 1 |
| Hot Plate | Electric with 3 heats | |
| Incubator | Oak heated by electric carbon lamp and provided with electrothermostat to be set at 37.5 C with cord and plug outside measurement approximately 12 x 9 x 9 | 1 |
| Jars (Staining) | Coplin | 3 |
| Labels | Miscellaneous | |
| Lens Paper | 1 package | |
| Litmus Paper | Red and blue (100 papers) | 2 |

middle shelves. To save time and labor solutions and apparatus in frequent use are grouped according to test. For example the *urinalysis shelf* beginning at the left should have the hydrometer the bottle of litmus paper the acetic acid for the albumin test the Benedict qualitative solution for the sugar test the Benedict quantitative solutions and the acetone testing reagents. Groups are arranged similarly for *gastric* and *duodenal* analyses for *blood chemistry* for *blood counts* for *stool examination* and for the *staining of smears*.

Care of Apparatus—The care of apparatus is of paramount importance. If bottles are promptly restored to the shelves and pipettes dishes and tubes immediately washed drained dried and returned to the proper places there will be a minimum of breakage contamination fumbling and expense.

Labels and Records—Solutions and specimens should be labeled accurately. As soon as the specimen is obtained the name of the patient and nature of the specimen are entered in a ledger book (p. 4050). On the completion of the examination the report is recorded. The information can then be transferred at leisure to the patient's medical record. Rubber stamps are convenient for this purpose.

The ledger functions as an entry book for charges. Even with minimal charges for laboratory procedure the office laboratory should soon prove self supporting and may even show a profit.

TABLE 176—SOLUTIONS, CHEMICALS AND APPARATUS READY FOR OFFICE LABORATORY
(continued)

| Article | Container | Amount |
|---|-----------------|---------|
| Volumetric Pipette (5 cc) | | |
| Evaporating Dish | | |
| Sodium Carbonate | Original | |
| Talc | Original | |
| Stirring Rod | | |
| Fermentation Tubes (2) | | |
| Lang's Reagent (p 3680) | Bottle | 120 cc |
| Ammonia | Original bottle | |
| Ferric Chloride (10 per cent) | Bottle | 120 cc |
| Obermeyer Reagent (p 3685) | Bottle | 1000 cc |
| Chloroform | Original bottle | |
| Tincture of Iodine | Bottle | 30 cc |
| Calcium Chloride (10 per cent) | Bottle | 120 cc |
| Ehrlich Reagent (p 1947) | Bottle | 120 cc |
| Benzidine | Original | |
| Hydrogen Peroxide | Original | |
| Colorimeter for P.S.P. | | |
| 10 per cent Sodium Hydroxide | Bottle | 1000 cc |
| <i>Blood Shelf</i> | | |
| Hemoglobinometer | | |
| N/10 Hydrochloric Acid | Bottle | 120 cc |
| Counting Chamber | | |
| Red Cell Pipette | | |
| White Cell Pipette | | |
| Glass Slides | | |
| 2 per cent Acetic Acid colored blue | Bottle | 30 cc |
| 3.8 per cent Sodium Citrate | Bottle | 30 cc |
| Distilled Water | | |
| Wright Stain | Original | |
| Cresyl Blue (1 per cent) | Original | |
| Sedimentation Tube (p 3708) | | |
| Blood Grouping Powders (p 3709) | | |
| Lithium Oxalate | Bottle | 30 gm |
| Blood Sugar Set (p 3715) | | |
| Sulfonamide Set (p 3717) | | |
| Serology Tubes | | |
| <i>Miscellaneous Shelf</i> | | |
| Antiformin | | |
| Carbolfuchsin Stain | Original | 30 cc |
| Acid Alcohol (10 per cent H_2SO_4 in 95 per cent alcohol) | Bottle | 120 cc |
| Loeffler Methylene Blue | Original | 30 cc |
| N/10 NaOH | Bottle | 1000 cc |
| Evaporating Dish and 1 cc graduated pipette | | |
| 1 per cent Alcoholic Phenolphthalein | Dropper bottle | 30 cc |
| Tüpfel Reagent (0.5 per cent dimethyl amino azobenzol) | Dropper bottle | 1000 cc |
| 25 per cent Magnesium Sulfate | Bottle | 30 cc |
| Sudan III | Original | 30 cc |
| Saturated Solution of Mercuric Chloride | Bottle | 1000 cc |
| Gram Stain (Crystal Violet in methyl alcohol) | Bottle | 30 cc |
| Pandy Solution | Original | 120 cc |
| Gram's Iodine | Bottle | 30 cc |
| Safranin | Original | 30 cc |
| Xylol | Original | |
| Immersion Oil | Original | |

The meatus is thoroughly cleansed with cotton soaked with an antiseptic such as bichloride of mercury 1:1000. The patient is then directed to void the first few ounces of urine into one vessel, a somewhat larger amount into a second vessel, and the remainder of the bladder contents into a third and larger bottle.

It has been assumed that the contents of the first vessel indicate the condition of the anterior urethra, of the second vessel the posterior urethra, and the third the bladder urine itself. Whether or not this hypothesis is correct, the last voiding is a close approximation of a catheterized specimen.

TABLE 177—ROUTINE ANALYSIS OF URINE

| Tests | Technic | Remarks |
|-----------------------------------|---------|---|
| Color | See | See p. 3666 |
| Transparency | | p. 3668 |
| Reaction | p. 3669 | Acidosis (p. 21) alkalosis (p. 72) |
| Specific Gravity | p. 3671 | Concentration and dilution tests (p. 3657) |
| Albumin | p. 3672 | Albuminuria (p. 230) |
| Bence-Jones Protein | p. 3673 | Amyloiduria (p. 303) |
| Sugar | p. 3674 | Glycosuria (p. 3676) |
| Microscopy of Urinary Sediment | p. 3680 | Pyuria (p. 2352) crystalluria (p. 3681) bacilluria (p. 3684) cylindruria (p. 369) spermatorrhea (p. 3694) ova and parasites (p. 3684) |

PRESERVATION OF THE URINE

If the specimen of urine cannot be examined while fresh, decomposition should be prevented. The specimen is best preserved in an icebox. The addition of sufficient *toluol* to cover the surface of the urine retards decomposition and does not interfere with any of the routine tests. Crystals of *thymol* may be used, but tend to interfere with tests for bile and protein.

ANALYSIS OF THE URINE

Routine analysis of the urine includes a general description of the color and transparency of the specimen, the determination of the reaction and specific gravity, tests for albumin and glucose, and microscopic study of the urinary sediment.

Additional tests which are performed when they are indicated are listed in Table 178.

Color (Routine).—The normal urine is *straw* or *amber colored*. The urinary pigments (urochrome, uroerythrin, and urobilin) are derived from

CHAPTER 166

LABORATORY METHODS THE URINE

THE urine is a highly complex fluid and a wide variety of factors are concerned with its secretion excretion and composition

Clinical urinalysis may afford information concerning

- 1 The state of *body hydration* (p 706)
- 2 The *functional capacity of the kidneys* (p 2240)
- 3 The occurrence and nature of *diseases of the kidney and urinary tract* (p 2334)
- 4 Disorders of *metabolism* (p 581)
- 5 Disturbances of the *cardiovascular system* (p 770)
- 6 Abnormalities of the *endocrine balance* (p 1144)
- 7 The *functional status of the liver* and the patency of the *biliary tract* (p 1985)
- 8 The equilibrium between *hemopoiesis* and *blood destruction* (p 1060)
- 9 The presence within the body of *drugs poisons parasites* and even *tumors* (p 2665)

COLLECTION OF THE URINE SPECIMEN

The Casual Specimen—For routine analysis the casual urine voiding is most frequently examined This is the least valuable specimen If there is any suspected abnormality a twenty four hour collection is requested

The Twenty four hour Specimen—For the collection of the twenty four hour specimen the patient discards the first morning voiding Thereafter the urines of that day and night up to and including the first voiding of the next morning are saved—preferably in a gallon bottle

If it is inconvenient for the patient to carry a large bottle he is instructed to mix and measure the total output and to fill an 8-ounce bottle from it and bring it to the office

Night and Morning Specimens—Separate night and morning specimens are collected when it is inconvenient for patients to obtain an entire twenty four hour voiding The last specimen at night and the first specimen of the morning are collected separately Nocturnal voidings are included with the morning specimen

Fractional Specimens—Fractional specimens are collected under special conditions e g for the *concentration and dilution tests* (p 3687) or in the control of *diabetic patients* (p 1216)

Catheterized Specimens—When formed elements are found in the specimen the site and nature of the pathological process must be determined To exclude contamination from the vagina or urethra a catheterized specimen is collected for microscopic and bacteriologic examination

Three Glass Test—In the presence of *pyuria* (p 2352) or *hematuria* (p 2306) it is sometimes inconvenient or inadvisable to perform catheterization An approximation of the catheterized specimen may be obtained by the three glass test

regulation of the hydrogen ion concentration of the plasma and contributes to the conservation of base

See *Acidosis* (p 721) *Alkalosis* (p 722)

The reaction of the urine is determined by the use of *indicator dyes* whose color depends on the pH. A commonly used indicator is *litmus paper* which is red in acid solution (pH 5.8) and blue when alkaline (pH 7.4). Slightly acid urine (pH 6.8) causes both red and blue litmus papers to turn reddish purple a color described as neutral

DIFFERENTIAL DIAGNOSIS OF

Deviations in Urinary Color

Deviations in urinary color may be due to alterations in concentration, the presence of incidental dyes or to admixture with blood, blood pigments or bile pigments. The last have important diagnostic significance

CAUSES

| | |
|----------------|--|
| Yellow | From <i>santonin</i> or <i>thymol</i> taken as anthelmintics (p 1834). Bile pigments with yellow foam on shaking. Confirm by chemical tests (p 3686). |
| Pink to Red | Discoloration of alkaline urine by <i>rhubarb senna</i> , <i>cascara</i> or <i>phenolphthalein</i> (p 1827). With blood or blood pigments. Demonstrate red cells by microscopy. Perform chemical test with <i>benzidine</i> (p 3685). |
| Green | From <i>thymol</i> taken as anthelmintic. Following use of <i>methylene blue</i> used as test dye (p 2473). Phenol poisoning causes urine to become green on standing (p 743). With bile pigments note foam on shaking. Confirm by chemical tests (p 3686). |
| Brown to Black | May follow use of <i>rhubarb senna</i> or <i>cascara</i> as cathartics (p 1827). With <i>melanotic tumors</i> black color develops on standing (p 3225). Bile pigments give foam on shaking. Confirm with chemical tests (p 3686). Blood and blood pigments may reveal red cells by microscopy. Chemical test with <i>benzidine</i> or absorption bands by spectrometry. |

Nitrazene paper (*phenaphthazine*) is a useful indicator in managing urinary infections. At pH 4.5 it is yellow at pH 6.0 it is gray green at pH 7.0 it is blue gray and at pH 8.0 it is blue.

Bromcresol purple (pH 5.2 to 6.8) and *methyl red* (pH 4.4 to 6.0) are more accurate indicators than litmus. With *bromcresol purple* a purple color shows that the urine is more alkaline than a pH of 6 while a red color with *methyl red* indicates an acid reaction greater than a pH of 5. To determine the hydrogen ion concentration of strongly alkaline urine the urine is tested with *phenol red* which is yellow when the pH is below 6.8 and turns purple red at a pH of 7.8.

the breakdown of hemoglobin. The depth of urinary color depends largely upon the water content. Dilute urine is pale and water clear. The concentrated urine is dark amber.

Transparency (Routine)—This varies from a limpid clarity (as in diabetes insipidus) to the most marked turbidity with a heavy sediment.

The opacity, which is due to the deposition of the various salts, disappears with heating and the addition of acid. An opacity that persists or is even intensified after heating and the addition of acid is due to the presence of formed elements such as *pus*, *blood* or *bacteria*. Rarely it may

TABLE 176—SPECIAL URINE TESTS

| Tests | Technic | Remarks |
|------------------------------|------------|---|
| Chemical | <i>See</i> | <i>See</i> |
| Acetone | p 3680 | Ketonuria (p 3680) |
| Diacetic Acid | p 3680 | Ketonuria (p 3680) |
| Indican | p 3685 | Indicanuria (p 3685) |
| Bile and Bile Pigments | p 3686 | Bilirubinuria (p 1954) |
| Urobilin and Urobilinogen | p 3686 | Jaundice (p 1954) hemolysis (p 1060) |
| Blood and Blood Pigments | p 3685 | Jaundice (p 1954) hemoglobinuria |
| Renal Function | | |
| Concentration | p 3687 | Renal function (p 2240) |
| Dilution | p 3687 | Renal function (p 2240) |
| Urea Clearance | | Renal function (p 2240) |
| Phenolsulfonphthalein | p 3688 | Renal function (p 2240) |
| Bacteriologic | | |
| Smears | p 3690 | Urinary infections (p 2334) |
| Cultures* | p 3690 | Urinary infections (p 2334) |
| Quantitative Chemical | | |
| Heavy Metals | | Poisonings* (p 743) |
| Salt* | p 1271 | Adrenal cortical insufficiency (p 1271) |
| Calcium* | p 1223 | Hyperparathyroidism (p 1223) |
| Sulfonamide Concentrations* | p 3690 | Sulfonamide therapy (p 88) |
| Pregnancy* | p 2619 | Diagnosis of pregnancy (p 2619) chorionepithelioma (p 2665) |

* Clinical Pathologist

be caused by fat globules (*chyluria*). Under any circumstance the sediment of the turbid urine is examined microscopically (p 3660).

It is advisable to filter a turbid urine before performing the tests for the detection of albumin and sugar.

Reaction (Routine)—Normal urine is usually *acid* in reaction, the pH approximating 6. The excretion of an excess of acid radicals tends to depress the pH to its lower limit (4.8). An excess of alkaline radicals (base) elevates the pH to the upper limits (8.2). By excreting a urine more acid or more alkaline the kidney plays an important part in the

RENAL

Nephropathoses

Parenchymatous nephritis with casts and good function (p 2362) Amyloidosis with hypos thenuria hepatosplenomegaly and positive Congo-red reaction (p 7) Focal interstitial nephritis with casts and normal renal function (p 2366) Diffuse acute glomerulitis particularly associated with deposition of sulfonamide crystals (p 2367) Diffuse vascular diseases such as generalized arteriosclerosis hypertension periarteritis nodosa with eosinophilia and acute diffuse lupus erythematosus with eruption (p 2372) Acute glomerulonephritis with hematuria oliguria high specific gravity and relatively normal renal function (p 2373) Chronic glomerulonephritis with occasional cast, increased volume decreased specific gravity and azotemia (p 2379) Nephrotic phases with decreased volume increased specific gravity marked edema hypoproteinemia and hypercholesterinemia (p 2383)

Poisonings

Irritation due to mercury phosphorus arsenic arsenamine lead bismuth turpentine carbon tetrachloride sulfonamides phenol salicylates ether or chloroform

Toxemia

Hypertensive toxemia of pregnancy (p 2638) or bilateral cortical necrosis (p 2330) Note elevation of blood pressure edema and convulsions Cholemas with jaundice or disturbances of liver function (p 1953)

Generalized Infection

Febrile disorders particularly bacteremias infectious hepatitis yellow fever malaria and subacute bacterial endocarditis Order blood cultures (p 50) Wassermann test icterus index (p 1947) and examine spreads for plasmodia (p 507)

POST RENAL

Obstructions

Infections involving urinary tract and producing obstruction hematuria hemoglobinuria or pyuria (p 2352) Refer to urologist for urography cystoscopy and catheterization of ureters Examine differential specimens by smear culture and carbol-fuchsin stain for acid fast bacilli (p 40) Inject into guinea pigs (p 63) Get biopsy where indicated Consider exploratory operation

Specific Gravity (Routine).—The specific gravity of urine is measured with the *hydrometer* or *urinometer*. To make the reading urine is placed in a wide test tube or a deep graduated cylinder. The urinometer is floated by a twirling motion in the urine specimen. When the urinometer comes to rest without impinging upon the bottom or sides of the receptacle the meniscus is read.

The specific gravity of normal urine approximates 1.020. There is a normal range of variation from 1.001 to 1.040.

As a general rule the specific gravity varies inversely with the volume

DIFFERENTIAL DIAGNOSIS OF

Albuminuria

Small quantities of albumin not detected by routine laboratory methods are found in renal urine. An increase in urinary albumin beyond 28 to 78 mg per liter constitutes a pathologic finding whose significance requires careful evaluation.

The proteins of the urine are chiefly albumin and globulin derived from circulating blood. The ratio of albumin to globulin is as 10:1. In most instances of renal disease predominance of albumin is due to the smaller size of the molecule which easily traverses glomerular endothelium when permeability is impaired. At times other proteins gain entrance to the urine as in hemoglobinuria and Bence Jones proteosuria.

Like azotemia albuminuria may be renal, prerenal or post renal. Post renal albuminuria is due to the presence of infection and invariably is associated with pyuria (p. 2352). Renal albuminuria is found in the nephropathies. Prerenal albuminuria accompanies systemic conditions in which there is neither renal damage nor infection.

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

PRERENAL

Physiologic

In the neonatal period. After a protein-rich meal. Following a cold bath or exposure to intense atmospheric cold. Following violent physical exertion.

Orthostatic

Albuminuria when patient assumes erect position. Usually in individuals of stotic habitus. Often associated with nephropathy. Unassociated with changes in renal function.

Circulatory

Forward failure associated with shock from whatever cause. Backward failure. Pressure on renal veins due to massive ascites, intra-abdominal tumors or huge spleen. Thrombosis of inferior vena cava.

Hemic

Severe anemia resulting in glomerular anoxemia and increased permeability of the kidneys. Obtain hemograms (p. 3704) and consider transfusion. Accompanying hemoglobinurias and hemoglobinurias.

Neurogenic

Following convulsions from whatever cause. No alteration in renal function.

Metabolic

Obstructive jaundice and conditions associated with cholemia. Note icterus and impairment of test of hepatic function (p. 1947). Hyperparathyroidism with diffuse cystic disease of bone. Thyrotoxicosis with elevation of BMR. Diabetes mellitus with nephrotic syndrome. Gout with tophi, podagra and hyperuricemia. Vitamin C deficiency with scurvy and therapeutic response to ascorbic acid.

The sample of urine is acidified with acetic acid. It is then diluted by the addition of water until the specific gravity is 1.008. The mixture is filtered and poured into the Esbach tube to the U mark. Esbach reagent (1 per cent picric acid in 2 per cent citric acid) is added to the R mark. The tube is closed with a rubber stopper and inverted several times. It is then set aside upright in a cool place. At the expiration of twenty-four hours the height of the precipitate is measured. A direct reading is made in grams of albumin per liter after correction for the amount of water used as a diluent.

BENCE JONES PROTEINURIA (ROUTINE)

The occurrence of Bence-Jones protein in the urine may be detected in the performance of the heat and acetic test for albumin. Bence Jones protein is a proteose of low molecular weight. Its presence is suspected if

DIFFERENTIAL DIAGNOSIS OF

Bence Jones Proteosuria

The diagnosis of Bence Jones proteosuria should not be difficult since the pathogenic mechanism is usually easily demonstrable.

CLINICAL MANIFESTATIONS AND DIAGNOSTIC FEATURES

| | |
|-----------------------|---|
| Multiple Myeloma | Bone malignancy characterized by painful infiltrations, multiple spontaneous fractures and characteristic cells in bone marrow (p. 1035). |
| Leukemia | Characteristic hemogram confirmed by marrow findings (p. 1035). |
| Chronic Nephropathies | Heavy albuminuria, elevation of blood pressure, hypoproteinemia and edema (p. 2362). Normal hemogram and marrow smear. |
| Idiopathic | In apparently healthy young persons with elevation of blood pressure. Normal bones, hemogram and marrow smears. |

the coagulum appears in the heated portion of the urine when the fluid is nearly tepid (40° to 50° C). The precipitate is heaviest at 60° C and disappears at the boiling point. As the urine cools to 60° C the precipitate reappears.

When Bence Jones protein and albumin are suspected to be simultaneously present in the urine, the specimen is boiled and filtered at the boiling point to remove the albumin. When the filtrate cools, the test for Bence-Jones protein is carried out.

GLYCOSURIA (ROUTINE)

Urine is tested for sugar by boiling a small quantity with an alkaline cupric sulfate solution (Benedict's qualitative solution).

Benedict's Qualitative Solution is prepared by dissolving 17.3 gm. cuprous sulfate in 100 cc. of distilled water. With the aid of heat, 100 gm. of anhydrous sodium carbonate and 173 gm. sodium citrate are dissolved in

of urine Following copious water drinking there is voided a large volume of urine with a low specific gravity (1 001) due to the excess of solvent over solutes When the intake of water is small or extrarenal water loss is increased (as in *vomiting diarrhea* or *sweating*) the volume is diminished and the specific gravity is elevated (1 030 to 1 040)

Despite the simplicity of measurements of urine volume and urine specific gravity these data furnish most valuable information concerning the hydration of the body and the functional state of the kidneys (p 2240)

THE DETECTION OF ALBUMINURIA (ROUTINE)

Technic—To detect the presence of an abnormal amount of albumin or protein in the urine an ordinary test tube (pyrex glass) is filled to within 1 inch of the top with the specimen that is to be examined Turbid urine should be filtered

The upper inch of the urine is brought to boiling over a small Bunsen flame The turbidity of the boiled portion is compared against a black background with that of the cool portion Two or three drops of 2 per cent acetic acid are then added The heated portion of the urine is again brought to the boiling point and the result is noted

Interpretation—No increase in the turbidity of the boiled portion before or after the addition of acid denotes a normal urine so far as the albumin content is concerned

Turbidity of the upper portion present before or after boiling but dissipated by the addition of acid indicates the presence of salts (phosphates carbonates)

Turbidity of the boiled portion which increases with heating and the addition of acid is due to a protein coagulum most likely an albumin The amount of albuminuria may be estimated roughly as varying from a faint cloud (1+) to a solid clot (4+)

The sediments of urine specimens containing albumin are examined microscopically

Acetic Acid and Merna Bodies—The addition of acetic acid to cold urine may produce a coagulum before heat is applied This reaction is given by proteins of uncertain identity (acetic acid bodies) A cloud (Merna bodies) may also form in the cold urine when acetic acid is added to a specimen that contains phenol

Orthostatic Albuminuria (Routine)—The orthostatic factor should be determined in every instance of albuminuria most particularly when the albuminuria is associated with a normal urinary sediment

On awakening in the morning and while still in the recumbent position the patient is requested to void into a specimen bottle Following this the subject arises remains upright for from twenty to thirty minutes and then urinates into a second bottle Each specimen is tested for albumin The orthostatic element is determined by a comparison of the relative amounts of albumin in the first and second voidings

Quantitative Tests for Albuminuria (Office Laboratory)—A rough quantitative estimation of the amount of albumin in the urine is performed by the method of Esbach For this purpose a special calibrated test tube is required (Esbach tube)

FERMENTATION TEST FOR SUGAR (ROUTINE)

Changes in the Benedict qualitative test may be due to the substances causing pseudoreduction or to the sugars (glucose lactose fructose pentose or galactose) Of all these substances only glucose is pathologically significant It may be recognized and identified by the fermentation reaction which requires the use of special tubes and fresh yeast

Technic.—The urine is boiled to kill bacteria After it has cooled a small pea sized lump of fresh yeast is added The mixture of urine and yeast is placed in the fermentation tube so that the solution fills completely the blind arm and displaces all gas or air bubbles The tube is incubated overnight at 37° C

In the presence of a fermentable sugar gas will replace the fluid at the blind end of the tube A control tube containing boiled urine but no yeast is observed for comparison Besides glucose only fructose is consistently fermented by yeast

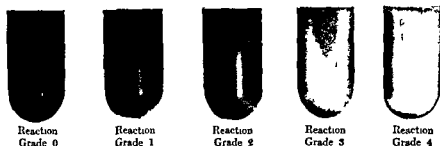


Fig 1069—Benedict's qualitative test for sugar in the urine The colored photographs above represent the appearance of solutions in test tubes containing 8 drops (0.4 cc) of urine added to 1 teaspoonful (5 cc.) of Benedict's qualitative solution the mixture being boiled for one minute over a free flame The urine added to the first tube on the left (Reaction grade 0) contained no sugar (dextrose) That added to the other tubes contained sugar (dextrose) in increasing concentrations from left to right Reaction grade 1 and reaction grade 2 represent reactions to traces of sugar (0.2 and 0.8 per cent, respectively) Reaction grade 3 and reaction grade 4 represent reactions with urine containing more than 1 per cent of sugar (1.5 and 4 per cent respectively)

QUANTITATIVE SUGAR TESTS (ROUTINE)

The *Benedict quantitative sugar test* is a refinement of the qualitative test It is based on the fact that a given quantity of glucose reduces a definite amount of copper in alkaline solution

The solution is prepared by dissolving crystallized sodium carbonate (200 gm) CP sodium citrate (200 gm) and potassium thiocyanate (125 gm) in 700 cc of distilled water Gentle heating is required To this is added a heated solution of pure crystallized copper sulfate (18 gm) in 100 cc of water Five cubic centimeters of 5 per cent potassium ferrocyanide are then added with stirring and the entire solution made up to 1000 cc with water

Modification of Benedict's Quantitative Test—Five cubic centimeters of Benedict's quantitative solution are placed in a porcelain evaporating dish

another 700 cc of water. The copper solution is added slowly to the alkaline solution and then made up to 1000 cc.

In the presence of a sugar containing a free aldehyde group (glucose, fructose, galactose, pentose, lactose) the cupric sulfate is reduced to cupric oxide, recognized by its characteristic yellow to brick red color.

BENEDICT'S TEST (ROUTINE)

Technic—Five cubic centimeters of Benedict's qualitative solution are heated to boiling in a clean test tube. Any change in the reagent on boiling indicates that it is imperfect or that the test tube is contaminated with a reducing substance.

If the boiled solution remains clear, 8 drops of urine are added. The mixture is boiled for two minutes and is then set aside to cool. The result is not read for at least fifteen minutes, since small quantities of sugar may cause a delayed reduction.

Interpretation—Normal—In the absence of reducing substance the reagent remains clear blue. The presence of a large amount of phosphate in the urine may produce a flocculent blue or grayish white precipitate, but the supernatant fluid is not changed.

Positive Reduction—The reduction of cupric sulfate to cupric oxide is indicated by an alteration in the color and clarity of the Benedict solution. The degree of change is proportional to the amount of sugar present. The test is capable of detecting quantities of sugar as small as 0.08 to 0.1 per cent, in which case the solution becomes turbid and pea green. A yellowish green color is produced when the urine contains about 0.5 per cent sugar. Loss of the greenish tint and the appearance of a yellow to brown precipitate indicates 1 per cent of sugar or more. Very large amounts of sugar will produce an orange to brick red test.

Pseudoreductions—In addition to sugars containing a free aldehyde group, several chemical groups may produce a slight and atypical reduction of cupric sulfate.

Glycuronates most often produce a pseudoreduction. The excretion of glycuronates is increased after ingestion of chloral hydrate, camphor, morphine, arsenic, antipyrine, chloroform, salicylic acid, amidopyrine, phenol, and sulfonal.

Urine containing *homogentisic acid*, as seen in *alkaptonuria*, produces a brown color when boiled with Benedict's solution.

Ascorbic acid will give a pseudopositive reaction in patients taking large amounts of pure vitamin C.

Highly concentrated urines, especially those of dehydrated patients, may give a positive reaction due to the presence of large amounts of *creatinine*.

The ingestion of *salicylates*, in addition to increasing the excretion of glycuronates in the urine, results in the excretion of *salicylic acid*, a substance which may produce a pseudopositive Benedict test. If *salicylic acid* is present, the addition of 10 cc of ferric chloride to 5 cc of urine produces a purple color.

FERMENTATION TEST FOR SUGAR (ROUTINE)

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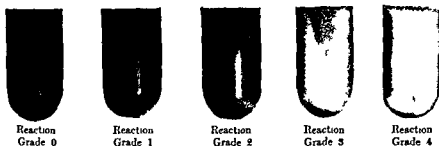


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Modification of Benedict's Quantitative Test—Five cubic centimeters of Benedict's quantitative solution are placed in a porcelain evaporating dish

DIFFERENTIAL DIAGNOSIS OF

*Disturbances Producing Reduction
of Benedict Solution*

The practitioner must not be led into the easy error of assuming that every person whose urine reduces Benedict solution is necessarily afflicted with diabetes mellitus. Rather should this diagnosis be made by exclusion after considering the factors enumerated in the Chart which follows.

| CAUSE | FERMENTATION BY YEAST | HYPERGLYCEMIA OR POSITIVE SUGAR TOLERANCE | DIAGNOSTIC FEATURES |
|-------------------------------|--------------------------|--|---|
| Alimentary | + | 0 | History of gluttony |
| Pharmacologic | + | 0 | Following anesthesia excessive doses of morphine or poisoning with chloroform. Pseudo-reduction due to salicylates, chloral hydrate, camphor, chloroform, or eucal, phenol, sulfonal, anipyrine, and amidopyrine. |
| Psychogenic | + | 0 | History of emotional upset. |
| Neurogenic | + | + | Evidences of increased intracranial tension due to head injury, neoplasm, or inflammation. Get neurologic status. Consider advisability of lumbar puncture. |
| Traumatic without Head Injury | + | 0 | In association with forward failure (shock) (p. 928). |
| Renal Glycosuria | + | 0 | Asymptomatic metabolic abnormality. Lowered renal threshold for sugar. Normal sugar tolerance and function. Requires no treatment other than reassurance. |
| Hyperthyroidism | + | + | Elevation of the basal metabolic rate. Note therapeutic response to iodide and Desacel (p. 1197). |
| Hyperpituitarism | + | + | Acromegaly or gigantism (p. 1156). |
| Pregnancy | + | + | Probably a manifestation of associated hyperpituitarism. Differentiate from lactosuria following delivery. |

| CAUSE | FERMENTATION BY YEAST | HYPERGLYCEMIA OF POSITIVE SUGAR TOLERANCE | DIAGNOSTIC FEATURES |
|-------------------|--------------------------|--|--|
| Diabetes Mellitus | + | + | Hypoinsulinemia associated with hyperglycemia and other metabolic disorders including acidosis and cachexia. etc. therapeutic response to diabetic control and insulin (p 1245) |
| Nephropathies | + | + | Probably an association of diabetes mellitus with a renal lesion |
| | + | 0 | Probably due to lowered renal threshold such as is responsible for the albuminuria (p 2370) |
| Alkaptonuria | 0 | 0 | Brown discoloration due to homogentisic acid. An asymptomatic metabolic anomaly |
| Cresatinuria | 0 | 0 | Pseudoreduction in dehydrated patients due to excess of cresatin in urine |
| Galactosuria | 0 | 0 | Following administration for liver function test (p 1947) |
| Fructosuria | + | 0 | May be associated with diabetes mellitus. Alimentary form in liver function test (p 1947). Essential fructosuria as a rare familial anomaly of no importance. Distinguish from glycosuria by polariscopy |
| Levulosuria | + | 0 | Same as fructosuria |
| Lactosuria | Occasional | 0 | Never during pregnancy. Associated with lactation. |
| Pentosuria | 0 | 0 | May be alimentary following large quantity of cherries, prunes, grapes and plums. Occasionally associated with diabetes mellitus (p 1246). May be congenital and familial anomaly of no importance |
| Sucrosuria | 0 | 0 | Cane sugar added by malingerer does not cause copper reduction |

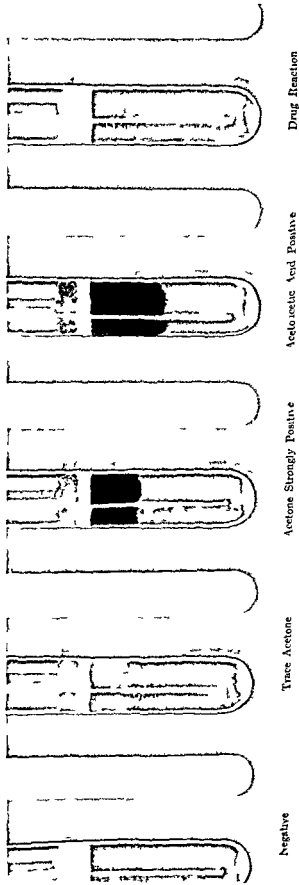


Fig 10 0—Sodium nitroprusside test for acetone and acetoacetic (diacetic) acid (Courtesy of Eli Lilly and Co.)

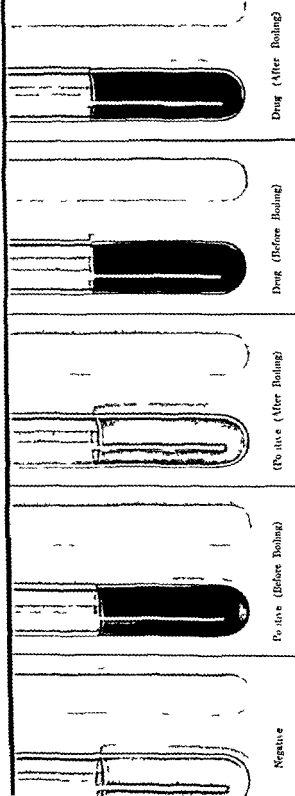


Fig 1071 —Gerhardt's ferric chloride test for acetic acid (Courtesy of J. H. Lilly and Co.)

(or a small beaker) Approximately 1 gm of anhydrous sodium carbonate and a little talc are added. The mixture is heated to boiling over a free flame. The urine is added drop by drop from a graduated pipette until the last trace of blue or green has disappeared from the boiling solution.

Calculation Five cubic centimeters of Benedict's solution are reduced by 0.01 gm of glucose. The percentage of sugar may be calculated by dividing 1 by the number of cubic centimeters of urine used. If 2 cc of urine were used the percentage of sugar present thus is $\frac{1}{2}$ (expressed as 0.5 per cent). If 0.25 cc was required the urine contains 4 per cent of sugar.

The total quantity of sugar present in the entire specimen may be calculated by simple multiplication, i.e.

$$\text{volume of urine in cc} \times \text{per cent of sugar} = \text{grams of sugar}$$

ACETONE AND DIACETIC ACID (KETONE BODIES) (OFFICE LABORATORY)

Though not routine tests the urine should be examined for acetone and diacetic acid in patients with *glycosuria* in the presence of *coma* in those who are on *ketogenic diets* (p 675) and when the *acidifying salts* are prescribed.

Nitroprusside Test for Acetone and Diacetic Acid (Rothera) (Office Laboratory)—A purple compound (ferropentacyanide) is formed by the interaction of ketones and nitroprusside.

Technic—Lang's reagent is prepared by dissolving 10 gm of sodium nitroprusside in 10 cc of glacial acetic acid. Water is added to make 100 cc. To test urine 5 drops of the reagent are added to 2 cc. The mixture is then overlaid with concentrated ammonia.

Interpretation—In the presence of acetone and diacetic acid a reddish purple ring forms at the junction of the fluids. The speed of development and the intensity of the color vary with the amount of ketones present. A positive test is recorded as +, ++, +++ or ++++.

This test is very delicate and will detect acetone in dilutions of 1 in 20,000 and acetoacetic acid in dilutions of 1 in 400,000.

Ferric Chloride Test for Diacetic Acid (Gerhardt) (Office Laboratory)—The interaction of acetoacetic acid and ferric chloride produces a deep red color.

A few drops of a 10 per cent aqueous solution of ferric chloride are added to 5 cc of freshly voided urine. A precipitate of ferric phosphate forms. As additional drops of solution are added a deep red color is produced in the presence of acetoacetic acid. This color fades on boiling. A positive test indicates acetoacetic acid in dilution of at least 1 in 1000.

False positive reactions are given by acetylsalicylic acid, antipyrine, cyanates or acetates. The color produced by these substances persists in urine which has been boiled for two to three minutes.

See *Acidosis* (p 721).

MICROSCOPY OF THE URINARY SEDIMENT (ROUTINE)

The routine urine examination is completed by a study of the sediment. After the urine has been permitted to stand in a special sedimenta-

tion glass the material for microscopic examination is obtained from the bottom of the vessel. A clean pipette stoppered by the index finger is introduced into the vessel. When the tip of the pipette reaches the bottom of the glass the finger is removed and urinary sediment enters the pipette from below. Before removing the pipette the upper end is again stoppered by the finger. The material collected in the pipette is transferred to a glass slide where it is examined under a coverslip.

A survey of the sediment is made with the low power objective of the microscope. The light should be cut down. With strong light it is

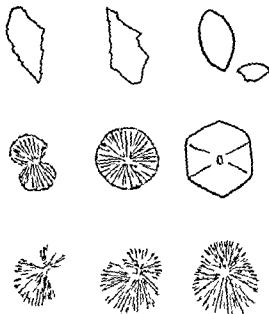


Fig. 102.—Crystals appearing in human urine after administration of *Sulfapyridine*—arrowheads and whetstones; *Sulfathiazole*—striated dumb-bells (shocks of wheat with central binding), rosettes with radial striations and renula; hexagonal platelets (all structures symmetrical); *Sulfadiazine*—striated dumb-bells (shocks of wheat with eccentric binding) and cell forms with radial striations (all structures asymmetrical) ($\times 40$).

difficult to recognize the formed elements of the urinary sediment particularly hyaline casts.

The high dry objective is useful in differentiating red blood cells from the white cells.

APPEARANCE OF URINARY SEDIMENT

The urinary sediment may contain (1) *extraneous material*, (2) *crystals* and *amorphous debris*, and (3) *organized structures*.

The *extraneous substances* are identified to avoid confusion with clinically important materials. These extra urinary contaminants include fibers

(or a small beaker) Approximately 1 gm of anhydrous sodium carbonate and a little talc are added The mixture is heated to boiling over a free flame The urine is added drop by drop from a graduated pipette until the last trace of blue or green has disappeared from the boiling solution

Calculation Five cubic centimeters of Benedict's solution are reduced by 0.01 gm of glucose The percentage of sugar may be calculated by dividing 1 by the number of cubic centimeters of urine used If 2 cc of urine were used the percentage of sugar present thus is $\frac{1}{2}$ (expressed as 0.5 per cent) If 0.25 cc was required the urine contains 4 per cent of sugar

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Cylindroids—Cylindroids are long filaments of mucus found in normal urine. They are not to be confused with casts. They tend to curl and twist and often have frayed out tapering ends in contrast to the smooth rounded ends of casts.

Red Blood Cells—Microscopy is the most sensitive method of detecting blood in the urine. Less than 10 cells per cu. mm. may be discovered microscopically. It requires the presence of 1000 erythrocytes per cu. mm. to give a positive chemical test (guaiac or benzidine).



Fig. 1074—Mucous threads in urine. These are often wrongly called cylindroids ($\times 350$).

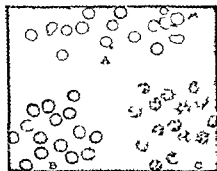


Fig. 1075—Red blood corpuscles in urine. A. Shallow cells from a case of nephritis. B. Fresh red corpuscles. C. Cremated corpuscles in a urine of high specific gravity ($\times 475$).

Grossly bloody urine has a red or brownish red color. A smaller quantity of blood produces a smoky tint. A macroscopically clear urine may contain isolated or even numerous red cells.

Red blood cells appear in fresh urine as round, refractile, biconcave disks with a yellowish green color. In old urine or in alkaline urine the cells crenate and lyse and appear as faint, colorless, irregular ghost cells. See *Differential Diagnosis of Hematuria* (p. 2306).

Fat oil droplets and **spherical forms** of urinary crystals resemble red cells. A drop of glacial acetic acid placed at the end of the coverslip

hair and oil globules the latter usually being derived from lubricants placed on catheters

Crystalline and amorphous sediments appear in both acid and alkaline urine. In the former may be recognized crystals of uric acid, amorphous sodium urate, calcium oxalate and the various crystalline forms of the sulfonamides (p 88). In the alkaline urine phosphates, calcium carbonate and ammonium biurates are deposited.

The recognition of the crystals assists in determining the chemical composition of *calculi* (p 2320) suggesting indications for dietotherapy.

The *organized structures* include the various types of casts, white and red blood cells, clumps of pus, epithelial cells, spermatozoa, bacteria, ova and parasites.

Casts—Casts are formed by the coagulation of protein within the tubules of the kidneys. They are classified according to their appearance into (1) *hyaline*, (2) *granular*, (3) *waxy* or (4) *organized*. A rare form of cast is composed of *calcium phosphate*. The presence of casts usually



Fig 1073—Hyaline and finely granular casts ($\times 350$) *

suggests *renal disease*. In the absence of albuminuria the finding of an occasional hyaline cast may be disregarded.

Hyaline Casts—Hyaline casts are transparent, refractile, tubular structures. They are best seen when illumination is cut down. Hyaline casts may be found in the urine of healthy people.

Granular Casts—Granular casts are seen in *acute* and *chronic nephritis*. They are never present in health.

Waxy Casts—Waxy casts are highly refractile and have a dull, opaque appearance. They are never found in normal urine and are most often seen in *chronic nephritis*. At one time they were believed diagnostic of amyloid nephrosis, but they occur in severe nephritis of any type.

Organized Casts—Organized casts consist of epithelial, red and white blood cells around a central hyaline core. They occur in *acute nephritis*.

Calcium Phosphate Casts—Calcium phosphate casts are hyaline casts covered with refractile granules. They are seen in patients with *hyperparathyroidism* and in other destructive lesions of bone in which the urinary calcium excretion is increased. They resemble granular casts but differ in that they are dissolved by dilute acetic acid.

* Todd and Sanford, Clinical Diagnosis by Laboratory Methods

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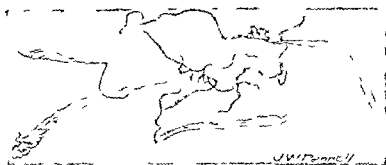


Fig 1074—Mucous threads in urine. These are often wrongly called cylindroids ($\times 350$)

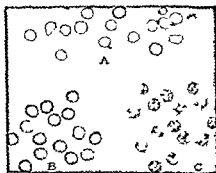


Fig 1075—Red blood corpuscles in urine. A Shadow cells from a case of nephritis. B Fresh red corpuscles. C Cremated corpuscles in a urine of high specific gravity ($\times 475$)

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Yeast oil droplets and spherical forms of urinary crystals resemble red cells. A drop of glacial acetic acid placed at the end of the coverslip

will lyse the red cells at the periphery of the slip but will not affect the other substances

Failure to find erythrocytes in the sediment of a red or brownish red urine suggests the presence of hemoglobin or its pigment derivatives See *Differential Diagnosis of Hemoglobinemias* (p 1074)

Pus Cells—The pus cells of urine have the appearance of the leukocytes of the blood It is rare to find more than an occasional white blood cell in a centrifuged sediment of a normal urine An increase in the number of white cells or the appearance of clumps warrants investigation

Conclusions regarding the significance of white blood cells in the urine of female patients should await the examination of a catheterized specimen of urine See *Differential Diagnosis of Pyuria* (p 2352)

Epithelial Cells—Epithelial cells are encountered in all urine specimens The female is apt to have considerable numbers The presence of epithelial cells in the absence of other cytologic finding has no pathological significance

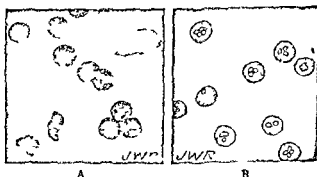


Fig 1076—Pus corpuscles in urine A As ordinarily seen At the lower left are two amoeboid corpuscles The large structure at the right is a bit of degenerated epithelium B When treated with acetic acid ($\times 475$) *

Spermatozoa—Spermatozoa are found in male urine after an emission Their presence in the absence of an emission requires study

BACTERIA

In alkaline urine motile bacteria of the colon group may be seen under the high power of the microscope A darkfield examination of a drop of urine sediment obtained from a patient with Weil's disease may disclose the presence of *Leptospira icterohaemorrhagiae* during the second week of the disease (p 360)

OVA AND PARASITES

The parasite that is most commonly encountered in urine is the protozoan flagellate *Trichomonas vaginalis* This organism is an actively motile pear shaped body about twice the size of a red blood cell (10 to 15 microns) At the anterior rounded end are four flagella of equal length

along one side is an undulating membrane which reaches to the middle of the body. The parasite moves fairly rapidly in the microscopic field and dives in and out of focus. The addition of a drop of Lugol's solution kills the organism and clearly reveals its structure. It is commonly encountered in female urine that has been contaminated by vaginal secretion. It may invade the urinary passages in either sex setting up a urethritis.

Individuals who are returning from regions where parasitic diseases are endemic may reveal in the urinary specimen the ova of the *schistosoma* with its characteristic lateral spine, the *microfilaria* of filariasis or the hooklets or scolices of the *Taenia echinococcus*.

INDICAN (OFFICE LABORATORY)

In testing for indicanuria 5 cc of urine and 5 cc of Obermeyer reagent are placed in a test tube. The reagent contains 10 per cent ferric chloride in concentrated hydrochloric acid. Two cubic centimeters of chloroform



Fig 1077.—Ova of *Schistosoma haematobium* with pus corpuscles in urine (photographs $\times 250$).

are added to the mixture. The tube is stoppered and inverted several times. It is then placed in a rack and observed.

The chloroform globules rapidly settle to the bottom. In the presence of indican these globules will be colored a blue or red, the intensity varying with the amount of indican present. Indicanuria is regarded by old clinicians as an evidence of intestinal auto-intoxication. It is often present in those who are presumably normal.

BLOOD AND BLOOD PIGMENTS (OFFICE LABORATORY)

Blood and blood pigments appear in the urine only under pathological conditions. The detection of red blood cells by microscopy has been previously described (p 3683). The chemical examination for the detection of the presence of blood and blood pigment is performed with benzidine.

Benzidine Test—The benzidine test is performed by preparing a saturated solution of benzidine in glacial acetic acid. Equal parts of hydro

gen peroxide are added to the benzidine test mixture. This mixture is layered on an equal volume of previously boiled urine. In the presence of blood or blood pigment a blue color appears at the zone of contact.

See *Differential Diagnosis of Hematuria* (p 2306), *Hemoglobinemia* (p 1074)

BILE AND BILE PIGMENTS (OFFICE LABORATORY)

Tests for Bile—The presence of bile in the urine produces a gross discoloration which may be yellow brownish green or black. With smaller quantities the pigment is observed in the foam produced by shaking the urine in a stoppered test tube. The discoloration is best noted by daylight or against a white background. It is seen with difficulty by artificial illumination.

For *chemical confirmation* of the gross presence of bile concentrated nitric acid is layered over 5 cc of urine in a test tube. A green or blue ring develops at the ring of contact (*Gmelin test*).

A more delicate test is performed with tincture of iodine diluted with an equal volume of distilled water. The dilute tincture is floated on the surface of the urine in a test tube. A green color indicates bile pigment, the iodine oxidizing bilirubin to biliverdin.

See *Differential Diagnosis of Obstructive Jaundice* (p 1954) *Jaundice in the Newborn* (p 2761)

Urobilin and Urobilinogen—Urobilin and urobilinogen are urinary pigments derived from bile and excreted into the intestinal tract. As bilirubin is reduced to stercobilin in the intestine a portion is absorbed and circulates in the blood as urobilinogen. Most of this is taken up by the liver and reenters the bile, but a small amount appears in the urine. If the liver cells are injured or if the excretion of bile pigment is excessive the amount of urobilin in the urine increases. If bile is excluded from the intestine no urobilinogen is formed and none is excreted in the feces and urine. The reappearance of urobilin in the urine may be an early sign of the subsidence of a complete biliary obstruction.

Test for Urobilinogen and Urobilin (Clinical Pathologist)—Before testing for urobilin and urobilinogen bile if present is removed by adding an equal volume of calcium chloride (10 per cent) to the urine and by filtering. The filtrate is used for the tests. Specimens of urine are diluted with water $\frac{1}{2}$, $\frac{1}{10}$, $\frac{1}{50}$ etc. A few drops of Ehrlich's reagent are added to each tube. The tubes are examined after ten minutes. A red tint indicates a positive test. The highest dilution in which the test is positive is determined. Normally this is up to $\frac{1}{10}$ dilution.

The absence of urobilinogen in undiluted urine indicates *complete biliary obstruction in jaundiced patients*.

The test for urobilin requires the use of 10 cc of urine freed from bile as described above. One drop of Lugol's solution and 10 cc of a saturated solution of zinc acetate are added. After filtration or centrifugation the presence of urobilin is indicated by a greenish fluorescence when the tube is viewed in bright sunlight against a black background.

See *Differential Diagnosis of Obstructive Jaundice* (p 1954) *Jaundice in Newborn* (p 2761) *Hemolytic Anemias* (p 1060)

URINARY TESTS OF RENAL FUNCTION (OFFICE LABORATORY)

The function of the kidney can be estimated accurately and with great ease. The data furnish an index of the gravity and course of renal disease.

The Concentration Test—The concentration test measures the ability of the kidney to reabsorb water and excrete solids as indicated by the maximum specific gravity of the urine after a moderate period of fluid deprivation.

Technic—The patient is instructed to drink nothing after his usual lunch on the day before the test. The evening meal should be complete but should be taken without fluid. The bladder is emptied before retiring and this voiding as well as all others during the night are discarded.

In the morning the first voided urine is kept in a separate bottle. Breakfast and fluids are withheld until the test is complete. At the end of each one hour period for three hours after the first morning voiding the bladder is emptied and the urine is kept in a separate bottle. Each bottle is labeled with the time when the specimen was obtained. The only examinations required are the measurements of volume and the estimation of specific gravity.

Results—If renal function is unimpaired the specific gravity of at least one specimen will exceed 1.022. The maximum specific gravity may reach 1.032. Dependent upon the degree of renal impairment the maximum specific gravity will be lowered until the fixation level (1.010) is reached in cases of severe renal insufficiency (p. 2275).

The highest specific gravity may be obtained in different specimens in different patients. Often the specific gravity of the first specimen is higher than the others despite the longer period of water restriction. In patients with *glycosuria* and *albuminuria* the specific gravity is corrected for the presence of these substances to eliminate false high values.

Interpretation—The limitation of concentrating power of the kidney is an early sensitive sign of impaired renal function. In *acute nephritis* this may be the only evidence of a functional defect while other tests (phenol sulfonphthalein and urea clearance) are normal. When the fixation level has been reached as in cases of *chronic nephritis* renal function has been reduced to at least 30 per cent of normal. To follow the further decrease in function other tests such as the urea clearance test must be used.

It is impossible to carry out a satisfactory concentration test in an *edematous patient* for the elimination of edema fluid may mask the ability to concentrate. Similarly a diet low in protein and sodium chloride may not yield enough urinary solids to cause a high specific gravity even with a small volume of urine.

As with other renal function tests the influence of extrarenal factors should be separated from those that are renal. Temporary impairment of concentrating power may be encountered in the presence of anatomic ally intact kidneys in cases of *congestive heart failure*, *peripheral circulatory collapse*, *severe anemias* and in some *endocrine disorders* (Addison's disease, diabetes insipidus).

The Dilution Test—The dilution test measures the ability of the kidney to excrete a large volume of water given by mouth.

Technic—For the urine dilution test the patient omits breakfast. The

gen peroxide are added to the benzidine test mixture. This mixture is layered on an equal volume of previously boiled urine. In the presence of blood or blood pigment a blue color appears at the zone of contact.

See *Differential Diagnosis of Hematuria* (p 2306) *Hemoglobinemia* (p 1074)

BILE AND BILE PIGMENTS (OFFICE LABORATORY)

Tests for Bile—The presence of bile in the urine produces a gross discoloration which may be yellow, brownish green or black. With smaller quantities the pigment is observed in the foam produced by shaking the urine in a stoppered test tube. The discoloration is best noted by daylight or against a white background. It is seen with difficulty by artificial illumination.

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A more delicate test is performed with tincture of iodine diluted with an equal volume of distilled water. The dilute tincture is floated on the surface of the urine in a test tube. A green color indicates bile pigment, the iodine oxidizing bilirubin to biliverdin.

See *Differential Diagnosis of Obstructive Jaundice* (p 1954) *Jaundice in the Newborn* (p 2761)

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See *Differential Diagnosis of Obstructive Jaundice* (p 1954) *Jaundice in Newborn* (p 2761) *Hemolytic Anemias* (p 1060)

cent sodium hydroxide is added to alkalinize and the volume is made up to 1000 cc. This is a 100 per cent standard color solution. Each of the diluted alkalinized specimens of urine is compared with the standard in a colorimeter.

An alternate method is to use the Dunning colorimeter* which consists of thirteen sealed ampules containing standard color solutions of different percentages. There is an open ampule for the unknown and a small box for comparison of the unknown and the standards. The reading is made directly in *per cent*.

Results—In normal persons the dye appears in the urine in from five to ten minutes after injection. Sixty per cent of the dye is eliminated in the first hour and about 75 to 80 per cent by the end of the second hour. *Decreased excretion* indicates renal insufficiency in the absence of extrarenal or postrenal modifying factors (*heart failure obstructive uropathies*). In general there is a rough parallelism between the degree of

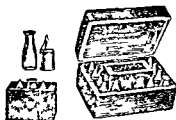


Fig. 1078—Dunning's colorimeter for the phenolsulfonephthalein test of kidney function.

renal insufficiency and the amount of dye excreted, as shown in the following table:

RELATIONSHIP OF RENAL FUNCTION AND PHENOLSULFONEPHTHALEIN EXCRETION†

| State of Renal Function | P.S.P. Excretion % |
|-------------------------|--------------------|
| Normal | 60 or over |
| Slight impairment | 59 to 40 |
| Moderate impairment | 39 to 25 |
| Marked impairment | 24 to 11 |
| Maximal impairment | 10 to 0 |

Failure to obtain all the urine during the collection period after the injection of dye will give a low value. Low values may also occur in *edematous patients in collapse* in patients taking *saline cathartics* in *anesthetized* or *heavily sedated patients* and *during pregnancy*. In general the sensitivity is less and is considerably lower than that of the concentration test. Normal values may be obtained in patients with impaired concentrating power.

Hynson, Westcott and Dunning

† Aft. Mosenthal and Lewis

bladder is emptied and 1200 cc of water are then drunk within ten to fifteen minutes (approximately 20 cc of water per kg of body weight). The urine is collected in a separate container at hourly intervals for four hours. The *volume* and *specific gravity* of each sample are determined. The patient should recline during the test as the rapidity of the water excretion is affected by posture being greater while recumbent.

Results—In the presence of *normal* renal function the hourly urine volume rises gradually to a peak at the end of the second hour and then slowly falls to reach basal levels during the fourth hour. The specific gravity of the urine is depressed to 1.001 to 1.002 at the height of the diuresis and tends to remain low. The total volume of urine voided during the four hour diuresis period usually equals or exceeds the volume of water administered.

In the presence of *renal impairment* the urine volume fails to increase in response to the administered water, the excretion of water is delayed and the urine specific gravity remains more or less fixed at 1.010.

Interpretation—The dilution test adds little to knowledge gained by the concentration test. If the specific gravity of the urine is not impaired the retarded excretion of water is usually the result of disturbances in the hormonal equilibrium and in the electrolyte balance. Water excretion of this type is seen in *dehydrated patients* after the administration of the pituitary antidiuretic factor in individuals suffering from *sodium chloride depletion*, in *adrenal insufficiency* and in some cases of *diabetes insipidus*.

In the presence of impaired concentrating power the defective water excretion is due in part to *impaired renal function*, to *electrolyte disturbances* resulting from renal insufficiency and to *attendant cardiovascular weakness*.

The dilution test has the disadvantages of causing nausea in some patients and a possible overloading of the circulation in patients with cardiac difficulty.

See *Differential Diagnosis of Polyuria* (p 2231) *Anuria and Oliguria* (p 2232) *Retention of Urine* (p 2264) *Azotemia* (p 2276) *Nephropathies* (p 2364).

The Phenolsulfonphthalein Test (Office Laboratory)—The ability of the kidney to excrete phenolsulfonphthalein injected intramuscularly affords a measure of renal efficiency.

Technic—Half an hour before the injection of the dye the patient drinks 2 glasses of water to stimulate urine formation. Just prior to the injection the bladder is emptied and the urine discarded. One cubic centimeter of phenolsulfonphthalein (6 mg) is injected intramuscularly in the deltoid or gluteal region. The patient is instructed to save all the urine for the next two hours and ten minutes. At the end of the first hour and ten minutes the bladder is emptied and the urine saved. The second specimen is collected in a separate vessel. The two specimens are examined individually for the amount of dye.

To determine the *amount of dye* present enough 10 per cent sodium hydroxide is added to each specimen to bring out the red color. The volume of each is then made up with water to 1000 cc. A standard is prepared by adding 1 cc of the dye to 800 cc of water sufficient 10 per

for the blood determination (p. 3717). A simple quantitative test is performed by placing a drop of urine containing sulfonamide on a piece of wood pulp paper that has been moistened with hydrochloric acid. If as little as 0.01 per cent of the drug is present, a yellow color appears which deepens to orange in higher concentrations.

See *Differential Diagnosis of Nephropathies* (p 2364) *Azotemia* (p 2276)

BACTERIOLOGIC EXAMINATION OF THE URINE

Smears (Office Laboratory)—Knowledge of the bacteriology of urine is particularly important since the introduction of the sulfonamides as chemotherapeutic agents

For the most part sufficient information may be gleaned from the examination of the stained smear of the urinary sediment. The catheterized specimen should be obtained in a centrifuge tube. Three microscopic slides are prepared by spreading loopfuls of the sediment in thick and thin smears. Of these slides one is stained with *methylene blue* one by the *Gram method* and the last by *carbolfuchsin* (p 52) for the



Fig 1079.—Tubercle bacilli in urinary sediment (\times 800)

laborious search for tubercle bacilli. From the first two stained smears information can be obtained concerning the probable nature of the infecting organism. In all likelihood a *gram negative* rod is a colon bacillus and a *gram positive* coccus a streptococcus or staphylococcus. A smear for tubercle bacilli should be examined for two hours before it is reported negative.

Cultures of the Urine (Clinical Pathologist)—Urine cultures reveal the definite nature of the organism causing the infection. The findings are checked with the data obtained from the smears. In suspected tuberculosis of the urinary system guinea pigs are injected with centrifuged urine and examined after eight weeks for evidence of tuberculosis.

Sulfonamide Concentration—The urinary concentrations of free and acetylated sulfonamides are calculated by methods similar to those used

TABLE 179.—ANALYSIS OF BLOOD (Continued)

| Test | Technic | Normal Values | Remarks |
|------------------------------|---------------|----------------------------------|--|
| Coagulation Time | See p 3706 | 8-15 minutes | See Hemorrhagic diatheses (p 1106) heparin (p 1030) |
| Fibrinogen Time | p 1950 | 15-20 seconds | Hemorrhagic diatheses (p 1106) vitamin K deficiency (p 1111) |
| Clot retraction | p 3706 | 1-24 hours | Hemorrhagic diatheses (p 1104) |
| Fragility of Red Cells | p 3706 | 0.44% to 0.30% saline | Hemolytic anemias (p 1060) |
| Peroxidase Stain | p 3707 | | Leukemia (p 1100) |
| Sedimentation Rate | p 3707 | 18 mm in 1 hour | Infections (p 35) |
| Hematocrit determination | p 3707 | Men 40-54 cc Women 37-47 cc | Hemoconcentration (p 3708) hemodilution (p 3708) |
| Capillary Fragility | p 3708 | Less than 10 petechiae | Hemorrhagic diatheses (p 1106) vitamin C deficiency (p 1120) |
| Plasma Volume | | 40-50 cc per kilo of body weight | Shock (p 928) polycythemia (p 1092) |
| Blood Volume* | | 70-80 cc per kilo of body weight | Shock (p 928) polycythemia (p 1091) |
| Blood Grouping and Matching* | p 3708 | | Transfusion (p 3778) paternity tests |
| Rh Factor | p 1067 | | Erythroblastosis foetalis (p 1067) |
| Dough Landsteiner Reaction | p 1075 | | Paroxysmal hemoglobinuria (p 1075) |
| Acid Hemolysis Test | p 1076 | | Nocturnal hemoglobinuria (p 1076) |
| Cold Agglutinins | p 3711 | | Blood watching (p 3708) |
| Bacteriology | p 45 | | Infections (p 35) |
| Serology* | p 50 | | Infections (p 35) |
| Blood Chemistry | p 3712 | | Table 183 |

Refer to Clinical Pathologist

COLLECTION OF BLOOD SPECIMENS

Capillary and venous specimens are used for most blood examinations. The hematologist examines additionally the bone marrow usually obtained by *sternal puncture* (p 1035)

CHAPTER 167

LABORATORY METHODS THE BLOOD

A VAST fund of information is available from examination of the blood. Many of the tests can be performed in the office laboratory but some must be referred to the clinical pathologist. All of them are listed in Table 179.

TABLE 179—ANALYSIS OF BLOOD

| Test | Technic | Normal Values | Remarks |
|------------------|---------------|--|--|
| Hemoglobin | See p 3694 | Men 14-16 gm per 100 cc Women 13-15 gm per 100 cc | See Anemia (p 1055) polycythemia (p 1093) |
| Red Cell Count | p 3696 | Men 4.6-6.2 million Women 4.2-5.4 million per cu mm | Anemia (p 1055) polycythemia (p 1093) |
| White Cell Count | p 3698 | 5000-10,000 per cu mm | Leukocytosis (p 1096) leukopenia (p 1042) agranulocytosis (p 1036) |
| Platelet Count | p 3699 | 150,000-400,000 per cu mm | Thrombocytopenia (p 1144) thrombocytosis (p 1118) |
| Blood Smears | p 3699 | Neutrophils 50-70% Lymphocytes 25-35% Monocytes 2-8% Eosinophils 1-4% Basophils 0.5-1% | Anisocytosis poikilocytosis microcytosis macrocytosis polychromatophilia leukocytosis (p 1096) lymphocytosis (p 1098) eosinophilia (p 512) basophilia (p 1094) agranulocytosis (p 1096) leukemia (p 1100) malaria (p 307) microfilariasis (p 3221) mononucleosis (p 466) |
| Color Index | p 3705 | 0.8 to 1.0 | Hypochromic anemia (p 1099) hyperchromic anemia (p 1077) |
| Reticulocytes | p 3705 | 0-2% | Hyperchromic anemia (p 1077) hemolytic anemia (p 1060) |
| Bleeding Time | p 3706 | 1-4 minutes | Hemorrhagic diatheses (p 1108) |

standard a hemoglobin pipette into which blood is drawn to a measured point a mixing tube in which the acid hematin is formed from the blood and dilute acid. Various hemoglobinometers are available alike in principle but modified by individual investigators and manufacturers. Some require dilution of the acid hematin solution until it matches a single standard (Sahlb method) others use a single dilution of the acid hematin solution and vary the standard (Newcomer Wintrobe Hadden Hausser). The practitioner is referred to the manufacturer's circular for the exact details concerning the hemoglobinometer he decides to purchase. The Sahlb method using the Hellge hemoglobinometer is simple and inexpensive. The instrument consists of permanent glass standards and a square mixing tube graduated in grams per 100 cc of blood and also in per cent. The hemoglobin concentration is determined as follows:

- 1 Pierce the finger tip to obtain a free flow of capillary blood
- 2 Draw the blood to the 20 cu mm mark in the hemoglobin pipette wipe excess from the tip
- 3 Blow the contents into the square tube containing decinormal hydrochloric acid (to the 10 mark on per cent scale)

TABLE 180 —HEMOGLOBIN STANDARDS IN DIFFERENT HEMOGLOBINOMETERS

| Instrument | Grams (100% Hgb) |
|------------------|------------------|
| Sahlb Hellge | 14.5 |
| Wintrobe | 14.5 |
| Dare | 16.0 |
| Newcomer | 16.0 |
| Sahlb (original) | 17.3 |

- 4 Rinse the pipette several times with the acid mixture to effect a quantitative transfer
- 5 Wait 5 minutes for the full color to develop and dilute the acid hematin with distilled water drop by drop until it has exactly the same color as the standard. The solution should be mixed with a glass rod after each addition
- 6 Readings are made in grams per 100 cc and in per cent

The method is accurate to within 5 per cent. Some of the common sources of error are (1) improper light (2) failure to allow the full color to develop (3) failure to fill the pipette (4) failure to make a proper dilution (5) dirty instruments (6) dirty diluting fluid

Although the determination of hemoglobin is a simple routine procedure it has been the source of more than a little confusion due to the differences in standards. Blood containing 100 per cent hemoglobin with one instrument might read 80 per cent with another.

Most hematologists today believe that the normal hemoglobin concentration lies between 15 and 16 gm per 100 cc for men and 13 and 15 gm in women.

Capillary Blood—To obtain capillary blood the skin of the finger tip or ear lobe is punctured with a Hagedorn needle or a blood lancet. In infants the big toe or the heel may be used. The puncture must be sufficiently deep to produce a free flow of blood. 'Milking' of the part dilutes the blood with tissue fluid.

Venous Blood—The antecubital veins are commonly used to obtain venous blood. Other sites of venipuncture are the vessels of the back of the hand and the external jugular vein. In infants the scalp veins and the internal jugular may be entered.

To prevent syncope the patient should recline during the performance of venipuncture. A tourniquet or blood pressure cuff is applied to the upper arm. The latter is distended to a point midway between systolic and diastolic pressures. The skin over the vein is cleaned with iodine and alcohol. The skin immediately below the vein is put on the stretch. The needle is inserted parallel to the vein with a gradual downward

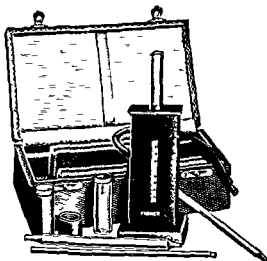


Fig. 1080—Sahli-Hellige hemometer

motion until blood appears in the syringe. When the required amount of blood has been obtained the blood pressure cuff is deflated. The needle is withdrawn. The patient is instructed to raise the arm over the head. A sterile sponge is applied with firm pressure to the puncture wound. This prevents the formation of an ugly ecchymosis.

The use of clean, dry syringes and needles prevents hemolysis and the clotting of blood in the syringe. Venous blood is prevented from clotting by a pinch of lithium oxalate. Plasma is obtained by centrifuging the oxalated blood and separating off the supernatant fluid. Serum is separated from clotted blood.

Except in bacteriologic work, blood need not be handled aseptically.

ESTIMATION OF HEMOGLOBIN (ROUTINE)

The hemoglobin concentration of the blood is easily and accurately determined by a simple colorimetric method whose basis is the conversion of hemoglobin to acid hematin. The test requires a *hemoglobinometer*.

standard a hemoglobin pipette into which blood is drawn to a measured point a mixing tube in which the acid hematin is formed from the blood and dilute acid. Various hemoglobinometers are available alike in principle but modified by individual investigators and manufacturers. Some require dilution of the acid hematin solution until it matches a single standard (Sahli method) others use a single dilution of the acid hematin solution and vary the standard (Newcomer Wintrobe Hadden Hausser). The practitioner is referred to the manufacturer's circular for the exact details concerning the hemoglobinometer he decides to purchase. The Sahli method using the Hellige hemoglobinometer is simple and inexpensive. The instrument consists of permanent glass standards and a square mixing tube graduated in grams per 100 cc of blood and also in per cent. The hemoglobin concentration is determined as follows:

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| Instrument | Grams (100% Hgb.) |
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| Wintrobe | 14.5 |
| Dare | 16.0 |
| Newcomer | 16.9 |
| Sahli (original) | 17.5 |

- 4 Rinse the pipette several times with the acid mixture to effect a quantitative transfer.
- 5 Wait 5 minutes for the full color to develop and dilute the acid hematin with distilled water drop by drop until it has exactly the same color as the standard. The solution should be mixed with a glass rod after each addition.
- 6 Readings are made in grams per 100 cc and in per cent.

The method is accurate to within 5 per cent. Some of the common sources of error are (1) improper light (2) failure to allow the full color to develop (3) failure to fill the pipette (4) failure to make a proper dilution (5) dirty instruments (6) dirty diluting fluid.

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Most hematologists today believe that the normal hemoglobin concentration lies between 15 and 16 gm per 100 cc for men and 13 and 15 gm in women.

ENUMERATION OF THE FORMED ELEMENTS OF THE BLOOD (ROUTINE)

The formed elements of the blood are the *red blood cells* or *erythrocytes* the *white blood cell* or *leukocytes* and the *platelets* or *thrombocytes*

Technic of the Blood Count—Blood counting requires the purchase of a *hemocytometer* a *red cell pipette* a *white cell pipette* a *cover slip* for the counting chamber, a *2 per cent solution of acetic acid* colored with *methylene blue* for the white count, and a *3.8 per cent solution of sodium citrate* for the red and platelet counts * For the latter freshly prepared solutions are required for accurate work

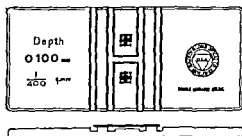


Fig 1081—An open counting chamber of excellent type. It is made in one piece and the surfaces of the platforms are highly polished making it possible to obtain Newton's bands as a criterion of proper application of the cover glass. The lower figure shows the chamber in cross section with cover glass in place.

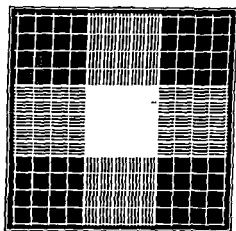


Fig 1082—Entire area of improved Neubauer ruling

The counting chamber in common use consists of a heavy glass slide with three narrow parallel platforms across the middle third. The center platform is 0.1 mm lower than the other two and has ruled upon it 2 areas of 9 sq mm divided into smaller squares. When the special cover glass is applied the chamber has a depth of 0.1 mm.

Each area of 9 sq mm is divided into nine squares of 1 mm each. These squares are further subdivided so the central square contains 400

CAUTION! In the purchase of the counting chambers and pipettes accept only those products that have been tested and approved by the United States Bureau of Standards.

small squares. With the improved Neubauer ruling now in common use the 400 small squares are separated by double lines into 25 groups of 16 each. The central millimeter containing the 400 small squares is employed in enumerating the red blood cells and platelets. Four of the square millimeters surrounding the central one are subdivided into 16 smaller squares

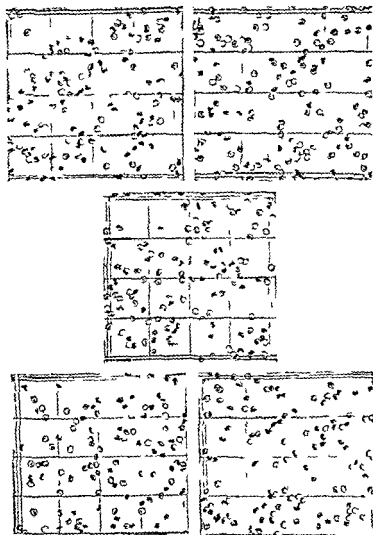


Fig 1083—Five fields sixteen squares each ($\times 430$)

These 4 sq. mm. at the corners of the ruled area are used in counting the white blood cells.

The bright line hemacytometer (Spencer Lens Co.) whose ruling is made on a metallic coating is a very satisfactory instrument.

Extreme attention to details is essential for accurate blood counts. Pipettes are useless if the ends are chipped. Unless they are perfectly dry, proper dilution and mixing are impossible. Other causes of error are failure to wipe excess blood from the tip of the pipette before diluting, failure to mix properly and to discard the first three drops from the pipette, air bubbles in the chamber, a flooded chamber and dirty fluid or apparatus, failure to obtain a free flow of blood and squeezing so that blood is diluted with tissue fluids.

The Red Count—For the red cell count the blood is drawn into the red cell pipette to the 0.5 mark. It is diluted to the 101 mark with 3.8 per cent sodium citrate solution. The pipette is well shaken with a lateral motion for three minutes. To effect the count the first few drops are discarded and a sample is placed on the counting chamber under the cover glass so that it covers the entire chamber. There must be no excess to bulge out or actually flow into the gutter at the side of the chamber. Allow three minutes for the cells to settle in the counting chamber before beginning the count.

Ordinarily the contents of 80 of the small squares are counted. The high dry objective of the microscope is used. To be assured of an equal mixture five areas of 16 squares each are counted (each corner of the square millimeter box and the center). The totals of each of these 16 squares should closely correspond when the mixture is uniform.

To obtain the number of the red blood cells per cubic millimeter of blood the sum of the cells enumerated in the 80 boxes is multiplied by 10 000, i.e. four zeros are added. See *Differential Diagnosis of Anemias* (p 1058) *Polycythemia* (p 1092).

The Platelet Count (Office Laboratory)—A rough estimation of the blood platelets can be obtained by surveying the elements in a well prepared smear. For the platelet count the same pipette and diluting fluid are used as for the red count. The sample of citrated blood is transferred to the counting chamber in the usual manner. At least twenty minutes must elapse to allow the platelets to settle. The light is cut down until platelets appear as small, highly refractile circular bodies. The contents of 80 small squares are counted. The estimation of the number of platelets per cubic millimeter of blood is obtained by adding four zeros, i.e. multiplying by 10 000. See *Differential Diagnosis of Thrombocytopenia* (p 1144) *Thrombocytosis* (p 1118).

The White Count (Routine)—In enumerating the leukocytes capillary blood is drawn up to the 0.5 mark in the white pipette. Two per cent acetic acid colored with methylene blue is drawn up to the numeral 11 etched at the top of this pipette. After waiting a few moments for the faking of the erythrocytes the pipette is shaken for three minutes. The first few drops are discarded and a sample is placed in the counting chamber as previously described. After a wait of two minutes to permit the cells to settle the numbers of leukocytes are counted in 4 of the larger squares. If the blood sample has been well distributed the totals of the cells in each of the 4 boxes should not vary by more than ten.

To calculate the number of white blood cells per cubic millimeter the total count for four squares is multiplied by 50.

See *Differential Diagnosis of Leukocytosis* (p 1097).

MORPHOLOGY OF THE STAINED BLOOD CELLS (ROUTINE)

After the blood is drawn for the red and white counts blood smear are made. A drop of blood is placed on a dry slide and spread with the edge of a second slide. The thickness and evenness of the smear vary with the size of the drop of blood and the speed with which it is spread. Considerable practice is required before good smears are consistently made. A good smear is essential for an accurate differential count.

Staining—The spread film is dried and fixed in the air. The surface is then flooded with Wright's stain. At the end of one minute 15 to 20 drops of distilled water are added. The diluted mixture is permitted to remain for four minutes while the slide is agitated gently. At the end of this time the stain is washed off with tap water. The slide is dried and it is ready to be examined directly with the oil immersion objective.

The length of time for flooding with the Wright stain will vary considerably with each preparation of the stain. The standard technic calls for one minute with the undiluted stain and four minutes with the diluted stain. The practitioner may find that he will get better results after flooding for two minutes and three minutes or some other variation. The Wright stain should be purchased in small 1 ounce bottles so that fresh samples are frequently available.

While there are a number of blood stains that are available for the expert it is generally agreed that the Wright stain suffices for routine clinical work. It is certainly recommended if only one blood stain is to be employed.

A good smear properly stained shows the red cells buff or orange in color separated from each other and not crenated. If they appear blue or purple the time of application of the dilute stain should be increased. The nuclei of the white blood cells are stained a deep blue.

The Appearance of the Normal Cells of the Peripheral Blood—The practitioner should content himself with the recognition of the normal cells of the peripheral blood. The positive identification of abnormal cells is beyond his province. If abnormal cells are present he may refer the patient directly to a hematologist or send the unstained blood slide to the expert for more accurate cytologic diagnosis. The normal stained film reveals red blood cells, white blood cells and platelets.

The Red Cells or Erythrocytes—The red blood cells stained by the Wright method are a golden orange. These are homogeneous disks with an average diameter of ~ 5 microns. The center of each cell appears somewhat paler than the periphery. The cells are of nearly uniform size and shape.

Sometimes intracellular parasites are seen (see *Malaria* p. 507). A platelet on a red cell may easily be confused with a malarial ring form.

Occasionally immature nucleated red cells (normoblasts, erythroblasts) are observed. These are usually reported as the number of nucleated red cells per 100 white cells. See *Differential Diagnosis of Anemias* (p. 1058). *Polycythemia* (p. 1022).

The White Cells or Leukocytes—The normal white cells are subdivided on the basis of their morphologic characteristics into (1) polymorphonuclear leukocytes, (2) lymphocytes and (3) monocytes.

Extreme attention to details is essential for accurate blood counts. Pipettes are useless if the ends are chipped. Unless they are perfectly dry, proper dilution and mixing are impossible. Other causes of error are failure to wipe excess blood from the tip of the pipette before diluting, failure to mix properly and to discard the first three drops from the pipette, air bubbles in the chamber, a flooded chamber and dirty fluid or apparatus, failure to obtain a free flow of blood and squeezing so that blood is diluted with tissue fluids.

The Red Count—For the red cell count the blood is drawn into the red cell pipette to the 0.5 mark. It is diluted to the 101 mark with 3.8 per cent sodium citrate solution. The pipette is well shaken with a lateral motion for three minutes. To effect the count the first few drops are discarded and a sample is placed on the counting chamber under the cover glass so that it covers the entire chamber. There must be no excess to bulge out or actually flow into the gutter at the side of the chamber. Allow three minutes for the cells to settle in the counting chamber before beginning the count.

Ordinarily the contents of 80 of the small squares are counted. The high dry objective of the microscope is used. To be assured of an equal mixture five areas of 16 squares each are counted (each corner of the square millimeter box and the center). The totals of each of these 16 squares should closely correspond when the mixture is uniform.

To obtain the number of the red blood cells per cubic millimeter of blood the sum of the cells enumerated in the 80 boxes is multiplied by 10 000 i.e. four zeros are added. See *Differential Diagnosis of Anemias* (p 1058) *Polycythemia* (p 1092).

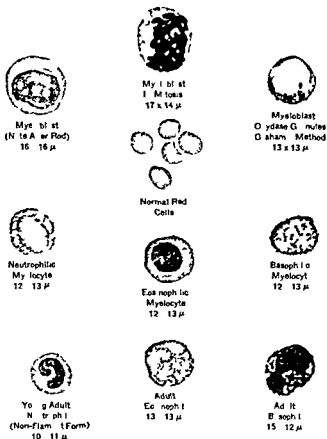
The Platelet Count (Office Laboratory)—A rough estimation of the blood platelets can be obtained by surveying the elements in a well prepared smear. For the platelet count the same pipette and diluting fluid are used as for the red count. The sample of citrated blood is transferred to the counting chamber in the usual manner. At least twenty minutes must elapse to allow the platelets to settle. The light is cut down until platelets appear as small highly refractile circular bodies. The contents of 80 small squares are counted. The estimation of the number of platelets per cubic millimeter of blood is obtained by adding four zeros i.e. multiplying by 10 000. See *Differential Diagnosis of Thrombocytopenia* (p 1144) *Thrombocytosis* (p 1118).

The White Count (Routine)—In enumerating the leukocytes capillary blood is drawn up to the 0.5 mark in the white pipette. Two per cent acetic acid colored with methylene blue is drawn up to the numeral 11 etched at the top of this pipette. After waiting a few moments for the taking of the erythrocytes the pipette is shaken for three minutes. The first few drops are discarded and a sample is placed in the counting chamber as previously described. After a wait of two minutes to permit the cells to settle the numbers of leukocytes are counted in 4 of the larger squares. If the blood sample has been well distributed the totals of the cells in each of the 4 boxes should not vary by more than ten.

To calculate the number of white blood cells per cubic millimeter the total count for four squares is multiplied by 50.

See *Differential Diagnosis of Leukocytosis* (p 1097).

diameter has a sharp cell outline and a nucleus separated into two or more lobes by a fine thread of chromatin. The nucleus stains a deep blue with



FURTHER STAGES OF NEUTROPHIL



FILAMENT FORM

Fig 1085—The white blood cells. The granular or myelocytic series. Wright stain (X 1000)

Wright's stain. The cytoplasm is barely visible and contains pale pink granules.

The neutrophils normally make up 50 to 70 per cent of the leukocytes.

Peppe and Farley: Practical Hematological Diagnosis

1 *Polymorphonuclear Leukocytes*—Polymorphonuclear leukocytes so named because of the lobed nucleus of the adult cell are further classi-

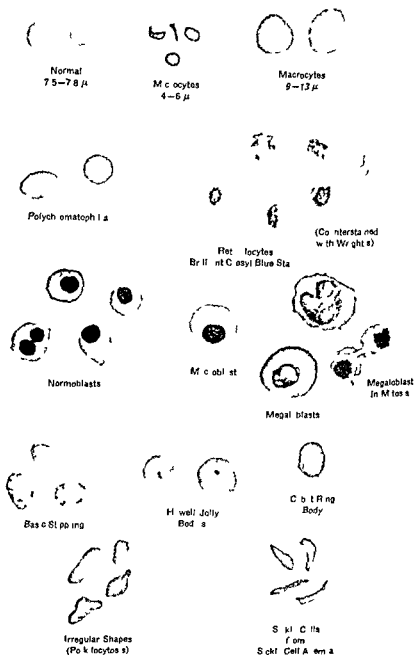


Fig 1084.—The red blood cells Wright's stain ($\times 1000$)

fied according to the staining of their cytoplasmic granules into *neutrophils*, *eosinophils* and *basophils*

NEUTROPHILS—The normal mature neutrophil is about 10 microns in

2 *Lymphocytes*—The lymphocytes are mononuclear round cells about 4 to 10 microns in diameter. They are usually slightly smaller than neutrophils. The typical lymphocyte contains a single sharply defined nucleus which stains a deep blue with Wright stain and a small amount of sky blue staining cytoplasm containing a few azurophilic granules. The nucleus is usually round but is sometimes indented at one side. The nuclear border










| Name of cell | Appearance of cell | Schilling | Arnet | Pons-Krumpholtz | Cooke-Ponder | Filament |
|--------------------------------|---|--------------|-----------|-----------------|--------------|--------------|
| Myeloblast |  | Myeloblast | Class I | Myeloblast | One lobe | Non filament |
| Premyelocyte |  | Premyelocyte | Class I | Premyelocyte | One lobe | Non filament |
| Myelocyte |  | Myelocyte | Class I | Myelocyte | One lobe | Non filament |
| Metamyelocyte |  | Juvenile | Class I | Metamyelocyte | One lobe | Non-filament |
| Young neutrophil |  | Band | Class I | Non-segmented | One lobe | Non-filament |
| Adult neutrophil (two lobe) |  | Segmenter | Class II | Segmented | Two lobes | Filament |
| Adult neutrophil (three lobes) |  | Segmenter | Class III | Segmented | Three lobes | Filament |
| Adult neutrophil (four lobe) |  | Segmenter | Class IV | Segmented | Four lobes | Filament |
| Adult neutrophil (five lobes) |  | Segmenter | Class V | Segmented | Five lobes | Filament |

Fig 1087—Schematic outline of all cells in the neutrophilic series showing their positions in the various classifications of neutrophilic immaturity

is sharp there is a narrow unstainable perinuclear clear zone. Indentation of the nucleus is a prominent feature of some lymphocytes seen in febrile states and blood dyscrasias (Türk's cells).

Large lymphocytes (12–15 microns in diameter) with pale nuclei and abundant cytoplasm are often encountered. These may have distorted

Johnson in Kracke and Parker: A Textbook of Clinical Pathology. The Williams and Wilkins Co.

See *Differential Diagnosis of Leukocytosis* (p 1097) and *Agranulocytosis* (p 1096)

EOSINOPHILS—Eosinophils (1 to 4 per cent of the polymorphonuclear cells) have eosinophilic granules which are large regular and bright red

LYMPHOCYTIC SERIES



Lymphoblast
16 x 16 μ



Tükürütan
Form
14 x 15 μ



Lymphoblast
Stained for
Cytochrome Granules
Graham's Method



14 x 13 μ



10 x 7 μ



8 x 7 μ



6 x 6 μ

Lymphocytes

MONOCYTIC SERIES



21 x 14 μ



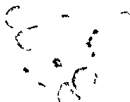
15 x 14 μ



13 x 12 μ



17 x 16 μ
(Macrophage)



Thrombocytes and
Red Cells

Fig 1056—The white blood cells Wright's stain ($\times 1000$)

The nucleus is usually indented and hour glass in shape The cell is somewhat larger than the neutrophil See *Differential Diagnosis of Eosinophilia* (p 542)

BASOPHILS—Basophils (0.5 to 1 per cent) are smaller than neutrophils and have numerous deep staining bluish black granules in the cytoplasm See *Differential Diagnosis of Basophilia* (p 1098)

be demonstrated by rimming a cover glass with petroleum jelly and placing this over a drop of blood on a slide. In two to twenty four hours if sickling is present there is a striking increase in the number of crescentic cells.

Microfilaria and Malarial Parasites—Microfilaria and the malarial parasites are recognized in the peripheral blood. The details are described elsewhere (pp 3321 and 507).

THE COLOR INDEX (ROUTINE)

This is a measure of the hemoglobin saturation of the red cell. It is calculated from the per cent of hemoglobin and the red cell count.

The hemoglobin percentage is used as the *numerator* in calculating the index. The *denominator* of the fraction is obtained by multiplying the two left hand figures of the red count by 2. Thus with a count of 5 000 000 and a hemoglobin of 100 per cent the color index would be $\frac{100}{100}$ or 1. The same red blood count with a hemoglobin of 50 per cent would give an index of $\frac{50}{100}$ or 0.5.



Fig 1089—Reticulated red corpuscles drawn from two slides stained as described in the text. The red corpuscles were yellowish green the reticulum and granules blue ($\times 1000$)

Indices less than unity are commonly seen in the secondary anemias. See *Hypochromic Anemia* (p 1089). Indices greater than unity occur in the *hyperchromic anemias* (p 1077).

THE RETICULOCYTE COUNT (OFFICE LABORATORY)

Normally there are less than 2 per cent of reticulocytes in the blood. When red blood cells are regenerating (as in a remission in the course of pernicious anemia) the number of reticulocytes rises considerably.

The reticulocyte count is made on the ordinary dry spread stained with a brilliant cresyl blue and the Wright's stain. A good sized drop of 1 per cent brilliant cresyl blue is allowed to dry on one end of a glass slide. A large drop of capillary blood is placed on the stained area and is mixed with the stain using a pin or edge of another slide. A thin smear is then made by the usual technic. The dry smear is counterstained with Wright's stain.

Reticulocytes contain a reticulum formed by the condensation of basophilic material. This substance is stained by the brilliant cresyl blue and

irregular nuclei containing many clear areas (unstained nucleoli) in *infectious mononucleosis* (p 466) See *Differential Diagnosis of Lymphocytosis* (p 1098)

3 Monocytes—The monocyte is the largest cell of the normal blood measuring 14 to 20 microns approximately two or three times the diameter of the red cell It contains a single deeply indented or horseshoe shaped nucleus which is commonly located to one side of the cell With Wright stain chromatin strands and clumps are readily recognized in these nuclei The cytoplasm around the nucleus is relatively wide It stains faintly and contains numerous fine azurophilic granules See *Differential Diagnosis of Monocytosis* (p 1099)

Platelets or Thrombocytes—Platelets are oval or irregular pieces of cytoplasm lilac colored by Wright's stain with a number of deep staining fine granules In the stained smear they appear in irregular clumps A rough estimate of the number of platelets in the blood is gained from the



Fig 1088—Blood in sickle-cell anemia active form stained film The diagnosis is best made from unstained wet preparations in which after a few hours the number of crescentic and stellate forms is greatly increased (Photograph about $\times 500$)*

smear See *Differential Diagnosis of Thrombocytopenia* (p 1144) and *Thrombocytosis* (p 1118)

Schilling Hemogram—In performing a differential white count the neutrophils may be classified according to their degree of maturity As originally indicated by Arneth the number of lobes of the nucleus is an index of the age of the granulocytes more lobes indicating greater maturity (*Arneth leukocyte index*) Schilling simplified this concept and widened its application by dividing the neutrophils into *myelocytes juvenile cells stab cells* and *segmented cells* An increase in the number of young cells (round indented and band like nuclei) is termed a shift to the left and represents a response of the bone marrow to an acute need for neutrophils (regenerative type) or to a toxic influence which prevents complete maturation (degenerative type)

Detection of Sickie Cells—Some Negroes and rare members of other races show a characteristic distortion of the red cells when oxygen is excluded from a cell suspension The presence of sickling may be noted by the crescent or oat shape of the corpuscles in stained smears It may

* Courtesy of V P Svedenstricker

PEROXIDASE STAIN (CLINICAL PATHOLOGIST)

In leukemia abnormal cells may be *lymphoid* or *myeloid* in origin. The peroxidase stain of Goodpasture sometimes helps in differentiation. Cells which give a positive peroxidase reaction and contain large blue cytoplasmic granules are polymorphonuclear neutrophils, eosinophils, myelocytes or myeloblasts. Monocytes and lymphoblasts occasionally contain granules but lymphocytes lack them entirely. See *Leukemia* (p 1100).

BLOOD SEDIMENTATION RATE (ROUTINE)

The rate at which the red cells settle in a specimen of citrated blood gives information of diagnostic and prognostic importance.

Linzenmeier Method—This simple method for determining the blood sedimentation rate requires specially calibrated test tubes. Each tube is etched at the 1 cc volume mark and at a point 18 mm below.

Technic—To perform the test 0.3 cc of 3.8 per cent sodium citrate are drawn up into a 2 cc syringe. The venous blood is then taken up into the syringe to the mark of 1.5 cc. The blood and citrate are mixed and transferred to the graduated test tube to the 1 cc mark. The test tube is placed in a holder which can be conveniently cut out of an ordinary cork. Observations are made every few moments to determine the exact time at which the red cells settle to the 18 mm mark. Normally this should take 1 hour or more.

Westergren Method—The sedimentation rate may also be estimated by measuring the number of millimeters of descent of the red cells in a given time. The citrated blood is placed in a special tube marked at the 200 mm height. The bottom end of the tube is sealed with putty, wax or clay, and the tube is set upright. At the end of an hour the descent of the red cells is measured. Normally the cells settle 0 to 15 mm in 1 hour in men and 0 to 20 mm in women. Results are recorded in terms of mm of descent at the end of 1 hour.

There is a certain amount of confusion amongst clinicians, some of whom record the sedimentation time in terms of the *duration of the fall* to a certain fixed point while others report the *number of millimeters* that the cells have descended in a given time. In recording the rate the method should be stated.

HEMATOCRIT READING (CLINICAL PATHOLOGIST)

Hematocrit readings accurately reflect changes in plasma volume and are of value in following the efficacy of therapy in cases in which there has been an excessive loss of body water or plasma such as in *shock* and *following burns*.

A number of hematocrit tubes requiring different amounts of blood are in use. The *Sanford Magath* tube requires 5 cc of blood and is highly satisfactory. The *Wintrobe* tube requires approximately 3 cc of blood. In performing hematocrit determinations the same anticoagulant should be used in making comparative readings. Potassium oxalate crystals cause the red cells to shrink.

appears as a network or wreath of threads. Five hundred red cells are noted and the per cent of reticulocytes is recorded by dividing the total number by 5.

BLEEDING TIME (OFFICE LABORATORY)

The bleeding time (*Duke method*) is determined by making a small cut in the lobe of the ear using a blood lancet or the point of a scalpel. At intervals of thirty seconds the blood is removed with blotting paper which should not touch the skin. No pressure is employed. The test is completed when blood flow spontaneously ceases.

An alternate method (*Ivy hemostasis bleeding time*) uses a blood pressure cuff placed around the upper arm and inflated to 40 mm. The flexor surface of the forearm (away from a vein) is punctured and the blood is wiped away from the bleeding spot with a piece of filter paper at intervals of 10 seconds until the bleeding stops.

The normal bleeding time is from 1 to 4 minutes by the Ivy method and 1.5 to 4 minutes by the Duke technique.

See *Differential Diagnosis of Commoner Bleeding Diatheses* (p 1119)

COAGULATION TIME (OFFICE LABORATORY) (LEE AND WHITE TEST TUBE METHOD)

To estimate coagulation time, 1 cc of blood obtained by venipuncture is placed in each of three clean, dry test tubes (10 x 100 mm). The tubes are placed upright in a rack. The first tube is gently tilted every thirty seconds until the blood is jelled sufficiently to permit inversion of the tube without the spilling of the blood. Then the second tube is tilted and finally the third. The time that it takes for the blood to clot in the third tube is the coagulation time.

See *Differential Diagnosis of Commoner Bleeding Diatheses* (p 1112)

CLOT RETRACTION (OFFICE LABORATORY)

The blood employed in the determination of the coagulation time is observed at room temperature. Normally, the clot should retract, leaving a clear layer of serum in about one hour. Clot retraction should be complete in twenty-four hours. Due to the prolonged coagulation time in *hemophilia* (p) marked separation of the red cells from the plasma may occur.

Retraction of the clot is closely related to the number of blood platelets present.

FRAGILITY OF RED CORPUSCLES (CLINICAL PATHOLOGIST)

The fragility of the red corpuscles is estimated by suspending them in varying concentrations of hypotonic saline solution. The normal cells settle, forming a red sediment at the bottom of the tube with a clear supernatant fluid above. Dissolution of the cells and hemolysis produces a uniformly brownish-red fluid with no sediment.

With normal blood, hemolysis begins in the tube containing 0.44 or 0.42 per cent saline solution. It is complete in the tube containing 0.34-0.28 per cent.

See *Differential Diagnosis of the Hemolytic Anemias* (p 1060)

Four principal blood types are recognized according to the demonstration of the factors *A B a* and *b*. The existence of three different classifications of blood types has produced considerable confusion. The Landsteiner classification of types *O A B* and *AB* is favored by the Health Committee of the League of Nations and is known as the International Classification. Until this grouping has become universally recognized the blood type should be reported with a statement of the system that has been employed.

Subgroups—The existence of subgroups of Type *A* is an occasional source of difficulty in typing and cross matching. The anomalous agglutinogens *A₁* and *A₂* and the agglutinins *a₁* and *a₂* may produce annoying reactions.

Approximately 18 per cent of *A* individuals are *A₁* or *A₂*; about the same percentage of *AB*s are *A₁ B* and *A₂ B*.

Agglutininogen M and N—In addition to agglutinogens *A* and *B*, human blood also contains agglutinogens *M* and *N* which are independent of the former and of no clinical significance since the corresponding agglutinins are rarely present in human serum. They are important however from a medicolegal standpoint and with the agglutinogens *A* and *B* give rise

TABLE 181—COMPARISON OF BLOOD GROUPS

| Per cent of Adults | International (Landsteiner) | Jansky | Moss | Red Cell Agglutinogens | Serum Agglutinins |
|--------------------|-----------------------------|------------|------------|------------------------|-------------------|
| 43 | <i>O</i> | <i>I</i> | <i>II</i> | <i>absent</i> | <i>ab</i> |
| 40 | <i>A</i> | <i>II</i> | <i>II</i> | <i>A</i> | <i>b</i> |
| 7 | <i>B</i> | <i>III</i> | <i>III</i> | <i>B</i> | <i>a</i> |
| 10 | <i>AB</i> | <i>IV</i> | <i>I</i> | <i>AB</i> | <i>absent</i> |

to 32 classes of individuals. Sera for the detection of agglutinogens *M* and *N* are prepared by immunizing rabbits with human cells containing these factors and are used in the investigation of paternity.

Agglutininogen Rh—The injection of the red blood cells of rhesus monkeys into rabbits or guinea pigs produces a serum containing an agglutinin which is different from *A B M* or *N*. *Agglutininogen Rh* is found in 85 per cent of the bloods of whites and is capable of producing *anti-rh agglutinins* on injection into an *Rh* negative individual. The relation of the *Rh* factor to pregnancy and *erythroblastosis foetalis* is discussed elsewhere (p 1067).

The injection of *Rh* positive blood into an *Rh* negative individual will result in the formation of *Rh* agglutinins by a process of isoimmunization. Subsequent transfusions with *Rh* positive blood in such an *Rh* negative individual will cause agglutination of the red blood cells and a reaction which may be fatal. This may occur despite careful accurate grouping and cross matching by the routine methods.

BLOOD TYPING

To determine the blood types the physician requires a red cell suspension of prospective donors and recipient and type *A* and type *B* serums.

Separation of the plasma from the formed elements of the blood is accomplished by centrifuging the whole blood to which an anticoagulant has been added. The blood is spun at high speed (2500 revolutions per second) for one half hour in a calibrated hematocrit tube. As a result the red blood cells are packed into the bottom of the tube. Atop the erythrocytes is a narrow layer of white cells and platelets. The remainder of the column is plasma. Under normal conditions the volume of the packed red cells is 40 to 50 per cent in males and 37 to 47 per cent in females. Greater packing occurs in hemoconcentration due to polycythemia or loss of plasma. Lesser packing is observed in anemias and hemodilution.

CAPILLARY FRAGILITY TEST (ROUTINE)

Capillary fragility is estimated by applying the blood pressure cuff to the arm. A circle, about the size of a quarter is drawn on the forearm just distal to the antecubital fossa. For a period of three minutes the



Fig. 1090.—Wintrobe hematocrit tube

tension in the cuff is maintained at a level midway between systolic and diastolic pressures. The number of petechiae are then counted in the circle.

Normally there should be less than ten lesions. More than twenty petechiae is definitely abnormal and indicates an *increase in capillary fragility* as observed in thrombocytopenic purpura (p. 1114). It also furnishes a preliminary test for the recognition of a *deficiency in vitamin C* as in subclinical scurvy (p. 1120).

BLOOD GROUPING

Blood typing and cross matching for the compatibility of blood donor and recipient are essential prerequisites to blood transfusion. Blood typing is also of value in determining paternity and in selecting donors for skin grafts. The tests are easily performed with minimum equipment.

Blood Types—Landsteiner (1900) first observed that the serum of a normal individual can cause the agglutination of the red blood cells of another normal individual. He demonstrated two *agglutinogenic substances* (*A* and *B*) and two agglutinins (*a* and *b*). The presence of an agglutinogen and its corresponding agglutinin in the same blood is incompatible with life; their admixture in a transfusion may be the cause of a severe and even fatal reaction.

agglutinogens of low sensitivity so that agglutination does not occur in the test but may occur when blood is transfused subtypings may not have been recognized or hemolysis may have occurred so that the visual phenomenon of agglutination is not observed

Cross Matching—Before every transfusion the donor's cells should be tested against the patient's serum for agglutination and hemolysis. A suspension of the donor's cells in saline (0.85 per cent) is prepared as in blood typing and a drop of the patient's serum is added. The mixture is stirred with a toothpick and examined for clumping under the low power of the microscope. The cross match should be done as close to 37° C as possible to prevent errors due to cold agglutinins (p 3711)

For transfusion a donor of the same type as the patient should be used if possible. If a universal donor (type O) has to be used a 1:10 dilu-

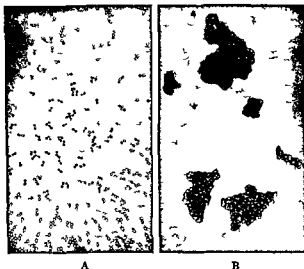


Fig 1091—Matching bloods for transfusion. *A* Corpuscles of a patient with serum of a prospective donor; no agglutination. *B* Serum of patient with corpuscles of prospective donor; strong agglutination. The blood of the donor is therefore unsuited for use in this case (photographs $\times 100$)

tion of the donor's serum should not cause clumping of the patient's cells in 30 minutes. During pregnancy it is important to determine whether or not the expectant mother is Rh negative or positive. A corresponding blood must be used to avoid a hemolytic reaction.

Cold Agglutinins and Autoagglutinins—The serums of certain individuals may contain agglutinins against their own red cells at temperatures from 0 to 5° C. Cold autohemagglutinins never cause reactions at body temperature and rarely at room temperature.

The detection of cold agglutinins against O cells and the patient's own cells is a simple confirmatory test for *virus pneumonitis* (p 2188). In this disease the cold agglutinin titer may rise as high as 1:20,000. The test is done by setting up a series of saline dilutions of the serum to be tested.

of high titer. The latter are commercially available in fluid and dry forms. They must be free of cold agglutinins (p 3711) and have a complement titer sufficiently lowered by storage so as not to produce hemolysis of the agglutinated cells. The type serums must be kept sterile since bacterial contamination impairs their usefulness.

By the Use of Type Serums—The actual typing is performed by mixing one drop of the saline cell suspension to one drop of type A serum on the left of a glass slide and one drop of B serum to the right of a clean glass slide. The commercial preparations of A serum are usually colored red while the B are green.

The mixture of cell suspension and type serum is carefully stirred with a clean toothpick and by gentle rotation of the slide. The final reading is made with the low power of the microscope after the two substances have been in contact for twenty minutes.

The readings are expressed in tabular form as follows:

| <i>Test Cells and A serum</i> | <i>Test Cells and B serum</i> | <i>Blood Type of Test Cells</i> |
|---------------------------------------|---------------------------------------|---------------------------------|
| No agglutination | No agglutination | O |
| No agglutination | Agglutination | A |
| Agglutination | No agglutination | B |
| Agglutination | Agglutination | AB |

By the Use of Cross Matching—When types A and B are not available the blood type may be determined by cross matching the cells and serum of a known type A or B individual and the cells and serum of the unknown as shown in the following schema:

I Known Blood Group A

Type A serum
+
Unknown cells

Type A cells
+
Unknown serum

Results

No agglutination
No agglutination
Agglutination
Agglutination

Agglutination
No agglutination
Agglutination
No agglutination

Interpretation Type

O
A
B
AB

II Known Blood Group B

Type B serum
+
Unknown cells

Type B cells
+
Unknown serum

Results

No agglutination
Agglutination
No agglutination
Agglutination

Agglutination
Agglutination
No agglutination
No agglutination

Interpretation Type

O
A
B
AB

Errors in Blood Grouping—Errors in blood grouping may arise for a variety of reasons. The serum may be of low titer having been permitted to deteriorate; the cell suspension may be too heavy; there may be present

TABLE 183 —BLOOD CHEMISTRY EXAMINATIONS

| Test <i>CB</i> = Cerebral Blood vol <i>S</i> = Serum | Normal Values per 100 cc | Remarks |
|---|-----------------------------|---|
| Sugar (<i>CB</i>) | 80-100 mg | See Hyperglycemia (p 733) hypoglycemia (p 734) diabetes mellitus (p 1216) pancreatic carcinoma (p 1913) |
| I protein (<i>S</i>) | 6-8 gm | Hypoproteinemia (p 706) nephrosis (p 2399) |
| Albumin (<i>S</i>) | 4-6 gm | Hypoproteinemia (p 706) |
| Globulin (<i>S</i>) | 1-2.5 gm | Hypoproteinemia (p 706) |
| Nonprotein Nitrogen (<i>CB</i>) | 2-55 mg | Azotemia (p 2270) renal insufficiency (p 2275) |
| Urea Nitrogen (<i>CB</i>) | 10-15 mg | Azotemia (p 2270) renal insufficiency (p 2275) |
| Uric Acid (<i>CB</i>) | 2-4 mg | Gout (p 2267) hyperuricemia (p 737) |
| Creatinine (<i>CB</i>) | 1-2 mg | Renal insufficiency (p 2275) |
| Cholesterol (<i>S</i>) Cholesterol Ester (<i>S</i>) | 140-190 mg 70-90 mg | Hypercholesterolemia (p 736) hypocholesterolemia (p 738) liver function (p 1917) lipodystosis (p 1907) thyroid disease (p 1197) cholelithiasis (p 1997) |
| Chloride (<i>S</i>) | 510-610 mg | Hypochloremia (p 73) hyperchloremia (p 732) |
| Sodium (<i>S</i>) | 320 mg | Hyponatremia (p 730) Hypernatremia (p 729) adrenal cortical deficiency (p 163) |
| Potassium (<i>S</i>) | 20 mg | Hypopotassemia (p 731) hyperpotassemia (p 731) familial paralysis (p 1416) adrenal cortical deficiency (p 163) |
| Calcium (<i>S</i>) | 9-11 mg | Hypocalcemia (p 723) hypercalcemia (p 723) parathyroid disturbances (p 1223) |
| Inorganic Phosphorus (<i>S</i>) | 3-4 mg | Hypophosphatemia (p 721) Hyperphosphatemia (p 723) parathyroid disturbances (p 1223) |
| Oxygen (<i>S</i>) | 12-14 vol (venous) | Anoxemia (p 3927) cyanosis |
| CO ₂ Content (<i>S</i>) CO ₂ Capacity (<i>S</i>) | 5-6 vol 60-70 vol | Acapnea (p 3830) hypercapnea (p 3930) acidosis (p 721) alkalosis (p 722) |
| Amylase (<i>S</i>) | Up to 320 units | Pancreatic disease (p 1913) |
| Phosphatase (alkaline) (<i>S</i>) | 2-9 Bodansky units | p 728 |
| Phosphatase (acid) (<i>S</i>) | 0-1.1 S.J.T. units | Cancer of prostate (p 2150) |
| Bilirubin (<i>S</i>) | 0.5 mg | Jaundice (p 1951) |
| Icteric Index (<i>S</i>) | | Jaundice (p 1951) |
| Van den Bergh (<i>S</i>) | | Jaundice (p 1951) |
| Sulfonamide (<i>CB</i>) | | Sulfonamide therapy (p 88) |
| Conjugated Phenols | | Renal insufficiency (p 275) |

from 1 5 to 1 20 000 after which the rack is placed in a refrigerator for twelve hours The presence of hemagglutination is detected by the magnification afforded by the concave substage mirror of the microscope The agglutination observed at 5° C completely disappears at 37° C Titers above 1 40 are abnormal and are seen in virus pneumonitis mumps orchitis hemolytic anemias and malaria These agglutinins may cause errors in blood typing and cross matching

TABLE 182—TECHNIC OF COLD AGGLUTININ TEST

| Tube No | Saline (cc) | Serum | Serum Dilution | 2% O Cells | Titer |
|---------|-------------|-----------------------|----------------|------------|--------|
| 1 | 0 8 | 0 2 cc | 1 5 | 0 2 | 1 7 |
| 2 | 0 5 | 0 5 cc of dilution 1 | 1 10 | 0 2 | 1 14 |
| 3 | 0 5 | 0 5 cc of dilution 2 | 1 20 | 0 2 | 1 28 |
| 4 | 0 5 | 0 5 cc of dilution 3 | 1 40 | 0 2 | 1 56 |
| 5 | 0 5 | 0 5 cc of dilution 4 | 1 80 | 0 2 | 1 112 |
| 6 | 0 5 | 0 5 cc of dilution 5 | 1 160 | 0 2 | 1 224 |
| 7 | 0 5 | 0 5 cc of dilution 6 | 1 320 | 0 2 | 1 448 |
| 8 | 0 5 | 0 5 cc of dilution 7 | 1 640 | 0 2 | 1 896 |
| 9 | 0 5 | 0 5 cc of dilution 8 | 1 1280 | 0 2 | 1 1792 |
| 10 | 0 5 | 0 5 cc of dilution 9 | 1 2500 | 0 2 | 1 3584 |
| 11 | 0 5 | 0 5 cc of dilution 10 | 1 5120 | 0 2 | 1 7168 |
| Control | | | — | 0 2 | — |
| 12 | 0 5 | 0 | | | |

CHEMICAL EXAMINATION OF THE BLOOD

In institutional practice where laboratory facilities are readily available the blood chemical values appear prominently on most charts Occasionally they furnish data of diagnostic and prognostic importance in the study of metabolic diseases More often they constitute luxuries and refinements which the general practitioner must eschew in favor of more easily available and less expensive forms of examination

The practitioner is required to keep informed of the progress of blood chemistry since it is only in this way that he may fully understand and elucidate the metabolic problems that confront him

COLLECTION OF BLOOD FOR CHEMICAL ANALYSIS

Specimens of blood for chemical analysis are usually drawn early in the morning after the patient has fasted for at least twelve hours If serum

- 5 *Alkaline Copper Solution* Dissolve 40 gm pure anhydrous sodium carbonate in 400 cc distilled water transfer to a liter volumetric flask Add 7.5 gm tartaric acid and 4.5 gm crystallized copper sulfate which have been dissolved in 100 cc of water with the aid of heat Mix and make up to one liter with distilled water
- 6 *Phosphomolybdic Acid Solution* Place 35 gm phosphomolybdic acid 5 gm sodium tungstate 200 cc 10 per cent sodium hydroxide and 200 cc distilled water in a liter beaker Boil vigorously for 20-40 minutes cool dilute to 350 cc with distilled water Add 125 cc of concentrated (85 per cent) phosphoric acid and dilute to 500 cc

Procedure

- 1 Measure 3 cc of oxalated blood into a 125 cc Erlenmeyer flask
- 2 Add 21 cc distilled water using portions to rinse the blood from the pipette
- 3 Add 3 cc of 10 per cent sodium tungstate solution and mix well



Fig 1093—Folin's tube for use in blood sugar estimations The narrow portion is 4 cm. long and 8 mm in diameter The bulb must be of such size that 4 cc of fluid will reach into the lower third of the constricted portion and not above it

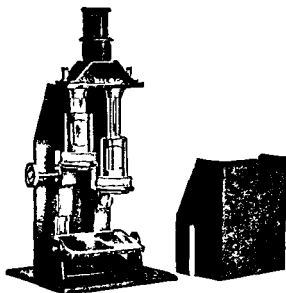
- 4 Slowly add one volume of sulfuric acid solution
- 5 Stopper with rubber and shake for 3 minutes
- 6 Loosen stopper and let stand for 10 minutes
- 7 Filter through ammonia free paper
- 8 Pipette 2 cc of filtrate into a Folin sugar tube
- 9 Prepare two standards Weak (100 mg) and strong (200 mg)
Use 1 cc of working standard (dextrose) for the weak and 2 cc for the strong Make up volume of weak to 2 cc
- 10 Add 2 cc alkaline copper solution to all tubes and place in boiling water for 6 minutes Remove all tubes at same time
- 11 Add 2 cc phosphomolybdate solution to all tubes
- 12 When tubes have reached room temperature dilute each with distilled water to 25 cc and mix well by inverting at least three times
- 13 Compare in colorimeter using standard that most nearly matches the unknown

is required the blood is taken into a centrifuge tube. If unclotted blood or plasma is to be employed a pinch of lithium oxalate is a useful anti-coagulant though sodium citrate, heparin and potassium oxalate may be substituted.

BLOOD SUGAR DETERMINATION

Because of the frequent necessity for blood sugar determinations the practitioner may wish to perform this procedure in his office laboratory.

The Folin Wu Macro Method.—The glucose in a protein free blood filtrate reduces an alkaline cupric to cuprous oxide. The precipitate is dissolved by a solution of phosphomolybdic acid forming a blue color the depth of the color is compared in a colorimeter (Duboseq) with a similar solution prepared from a standard amount of glucose.



Fog 109^a—The biologic colorimeter, a small colorimeter of the Duboseq type which is very satisfactory for clinical laboratory work. The light shield has been removed to show the cups.*

Solutions

- 1 Ten per cent *Sodium Tungstate* in distilled water salt should be arsenic and carbonate free
- 2 *Sulfuric Acid Solution* Prepare by adding 19 cc of concentrated acid (94 per cent) with a specific gravity of 1.835 to distilled water to make one liter
- 3 *Dextrose Standard (Stock)* Dissolve one gram of anhydrous dextrose (reagent grade) in 50 cc of benzoic acid solution (0.25%) and make up to 100 cc in a volumetric flask with benzoic acid solution
- 4 *Dextrose Standard (Working)* Transfer 2 cc of stock solution to a 100 cc volumetric flask and make up to mark with benzoic acid (0.25 per cent) so that 1 cc = 0.2 mg

- 5 *Alkaline Copper Solution* Dissolve 40 gm pure anhydrous sodium carbonate in 400 cc distilled water transfer to a liter volumetric flask Add 7.5 gm tartaric acid and 4.5 gm crystallized copper sulfate which have been dissolved in 100 cc of water with the aid of heat Mix and make up to one liter with distilled water
- 6 *Phosphomolybdic Acid Solution* Place 30 gm phosphomolybdic acid 5 gm sodium tungstate 200 cc 10 per cent sodium hydroxide and 200 cc distilled water in a liter beaker Boil vigorously for 20-40 minutes cool dilute to 350 cc with distilled water Add 125 cc of concentrated (85 per cent) phosphoric acid and dilute to 500 cc

Procedure

- 1 Measure 3 cc of oxalated blood into a 125 cc Erlenmeyer flask
- 2 Add 21 cc distilled water using portions to rinse the blood from the pipette
- 3 Add 3 cc of 10 per cent sodium tungstate solution and mix well



Fig 1093—Folin's tube for use in blood-sugar estimations. The narrow portion is 4 cm. long and 8 mm. in diameter. The bulb must be of such size that 4 cc. of fluid will reach into the lower third of the constricted portion and not above it.

- 4 Slowly add one volume of sulfuric acid solution
- 5 Stopper with rubber and shake for 3 minutes
- 6 Loosen stopper and let stand for 10 minutes
- 7 Filter through ammonia free paper
- 8 Pipette 2 cc of filtrate into a Folin sugar tube
- 9 Prepare two standards Weak (100 mg) and strong (200 mg)
Use 1 cc of working standard (dextrose) for the weak and 2 cc for the strong Make up volume of weak to 2 cc
- 10 Add 2 cc alkaline copper solution to all tubes and place in boiling water for 6 minutes Remove all tubes at same time
- 11 Add 2 cc phosphomolybdate solution to all tubes
- 12 When tubes have reached room temperature dilute each with distilled water to 25 cc and mix well by inverting at least three times
- 13 Compare in colorimeter using standard that most nearly matches the unknown

Calculations

$$\text{Weak standard } \frac{S}{u} \times 0.2 \times \frac{100}{0.2} = \text{mg per 100 cc}$$

$$\text{Strong standard } \frac{S}{u} \times 0.4 \times \frac{100}{0.2} = \text{mg per 100 cc}$$

If result is over 400 mg per 100 cc repeat using smaller amounts of filtrate (2 cc of filtrate = 450 mg glucose)

Glucose Tolerance Test (Office Laboratory)—The glucose tolerance test measures the ability of the tissues to store glucose after the administration of a large amount of the substance orally. The test is based on the height and duration of the elevation of blood sugar which follows the ingestion of a standard dose of glucose.

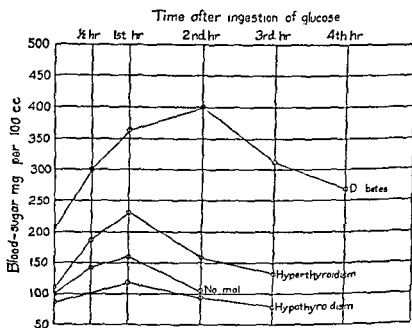


Fig 1094—Chart showing blood sugar curves obtained in the tolerance test

Technic—To perform this test the patient arrives in the morning, after having fasted for twelve hours. An amount of glucose equivalent to 1.75 gm for every kilogram (2.2 pounds) of body weight is weighed out and dissolved in water (4 gm per 10 cc). The solution is made more palatable by adding the juice of one lemon. The bladder is emptied and the urine specimen saved. The fasting blood sugar is obtained. The glucose solution is then drunk. Specimens of blood and urine are obtained 30 minutes, 60 minutes and 120 minutes after the glucose is taken. The patient rests in a reclining position until the test is completed.

Results—Normally the blood sugar level rises to 140 or 160 mg per 100 cc at the end of the first half hour. It usually returns to 110 mg per 100 cc at the end of two hours. A specimen taken at the end of three hours is usually below normal (80 to 90 mg).

A peak value of over 160 mg per 100 cc is abnormal. Abnormalities of the blood sugar curve consist of a flat (hypoglycemic response) or a high prolonged curve (hyperglycemic response).

In some instances when a flat curve is obtained the factor of delayed absorption should be ruled out by an *intravenous glucose tolerance test*. Twenty grams of 50 per cent glucose are injected intravenously and samples of blood are taken at one half one and two hours.

SIMPLIFIED PROCEDURE FOR DETERMINING SULFONAMIDES (O.L.)

A simplified procedure for determining the sulfonamide concentration of the blood can be performed in the office laboratory or at the bedside. It compares favorably in accuracy with methods in use by hospital laboratories.

Procedure*

Preparation of Materials—(1) A 10 per cent aqueous solution of sodium nitrite is poured over sheets of filter paper in a beaker. The papers are removed and allowed to dry; they are cut into strips (5 mm wide) and are stored in a wide mouthed amber bottle. (2) An aqueous solution of 1 per cent N(1 Naphthyl) Ethylenediamine Dihydrochloride is absorbed on filter paper using the same technique for nitrite paper. The strips are cut 1 cm in width. (3) A standard is prepared from tablets of sulfonamide used for oral administration as follows: A 7.5 gram (0.5 gm) tablet is dissolved in 950 cc of water and diluted to one liter. After shaking transfer 10 cc of this stock solution to a 100 cc flask and dilute to 100 cc (a slight turbidity due to starch does not interfere). (4) Make a solution of acid alcohol (1 cc concentrated HCl in 11 cc of 95 per cent alcohol).

Preparation of Filtrate—Measure 27 cc of 95 per cent alcohol (ethyl) into a 125 cc flask; add exactly 3 cc of ovalated whole blood. Stopper flask; shake well and filter into a tube (cover funnel to prevent evaporation).

Performance of Test (Using Large Tubes)—To 9 cc of alcohol and 1 cc of standard and to 10 cc of unknown (alcoholic filtrate) add 1 cc of acid alcohol. Shake. Add small piece of nitrite paper (3 x 5 mm) to each tube. Shake for 3 minutes. Then add one piece of dye paper (10 x 25 mm) and shake for 10 minutes. Compare colors of unknown and standard in tubes of equal size (5 cc of each). If unknown is darker dilute with alcohol noting amount added to match standard. If unknown is lighter dilute the standard.

Calculation

1. If the standard and unknown are equal without dilution the unknown contains 5 mg of sulfonamide per 100 cc of blood.
2. If the unknown was darker than the standard the number of cc of alcohol required for dilution plus 5 equals mg of sulfonamide per 100 cc.
3. If the unknown was lighter than the standard

$$\text{mg per 100 cc} = \frac{25}{\text{cc of alcohol} + 5}$$

If HCl is not available N/10 solution of H_2SO_4 may be used. It is not necessary to use chemically pure acid or distilled water if a blank is run on the reagents.

It is not necessary to use a separate standard solution for each sulfonamide; any one drug may be used as a standard and the result multiplied by the conversion factor.

TABLE 184—CONVERSION FACTORS FOR SULFONAMIDES

| Drugs Used as a Standard | Drug To Be Determined | | | | | | |
|--------------------------|-----------------------|---------------|-------------------|---------------|---------------|--------------|---------------|
| | Sulfanilamide | Sulfacetamide | Sulfanylguanidine | Sulfapyridine | Sulfathiazole | Sulfadiazine | Sulfasuxidine |
| Sulfanilamide | 1.00 | 1.24 | 1.24 | 1.45 | 1.48 | 1.45 | 2.06 |
| Sulfacetamide | 0.80 | 1.00 | 1.00 | 1.16 | 1.19 | 1.17 | 1.66 |
| Sulfanylguanidine | 0.80 | 1.00 | 1.00 | 1.16 | 1.19 | 1.17 | 1.66 |
| Sulfapyridine | 0.69 | 0.86 | 0.86 | 1.00 | 1.02 | 1.00 | 1.45 |
| Sulfadiazine | 0.69 | 0.86 | 0.86 | 1.00 | 1.02 | 1.00 | 1.42 |
| Sulfathiazole | 0.48 | 0.60 | 0.60 | 0.70 | 1.00 | 0.70 | 1.00 |

* Chemical determination is made by any method and the final result (mg. of sulfonamide per 100 cc.) is multiplied by the conversion factor.

CHAPTER 168

LABORATORY METHODS SPUTUM GASTRO INTESTINAL CONTENTS, DUODENAL CONTENTS THE STOOL

THE SPUTUM

SPUTUM is material that is obtained from the bronchi and alveoli. Saliva and nasopharyngeal secretions are sputum contaminants.

COLLECTION OF SPUTUM

Before collecting sputum the nose is cleared of nasal secretion. The contents of the nasopharynx are hawked into the pharynx and expectorated. The mouth is then rinsed. Only then may uncontaminated sputum be obtained.

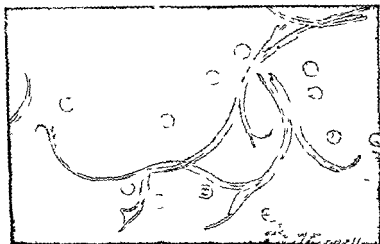


FIG. 169 —Elastic fibers in tuberculous sputum unstained as seen with a high power objective ($\times 400$)

When the sputum specimen is scant the pharynx is stimulated mechanically with a tongue blade. Sputum from a productive cough is then collected on a sterile swab inserted in the back of the pharynx. This procedure is usually necessary for the collection of specimens of sputum in infancy and childhood. It may also be employed in the early stages of pneumonia when there is not enough secretion for a gross specimen. The use of *cough plates* (p. 280) is yet another device particularly useful in influenza and pertussis. Gastric content and stool specimens may be examined for tubercle bacilli in patients particularly children who swallow sputum.

Sputum is collected in a covered container. Cardboard cups are burned. Glass containers are sterilized by prolonged boiling.

ROUTINE SPUTUM EXAMINATION

TABLE 185—MACROSCOPIC EXAMINATION OF SPUTUM

| Sputum | Integration |
|------------------------------|---|
| Excessive Quantity | <i>See</i> Bronchiectasis (p 2059) cavernous tuberculosis (p 2199) |
| Red streaking | Incidental bleeding from upper respiratory passages incipient tuberculosis (p 2199) |
| Red admixture | Lobar pneumonia (p 2171) pneumonitis (p 2185) chronic pneumonic infiltrations particularly tuberculous (p 2199) |
| Rusty | Lobar pneumonia (p 2171) |
| Prune juice | Pulmonary congestion (p 2086) |
| Brown to black | Retracted pulmonary congestion (p 2086) pneumoconiosis (p 2065) |
| Creamy yellow or green | Pulmonary infection (p 2214) |
| Layer formation | Bronchiectasis (p 2059) cavernous tuberculosis (p 2199) lung abscess (p 2214) |
| Putrid | Lung abscess (p 2214) |
| Macroscopic caseous material | Pulmonary tissue destruction as in caseous tuberculosis (p 2199) |
| Bronchial casts | Fibrinous bronchitis (p 2170) |

TABLE 186—MICROSCOPIC EXAMINATION OF SPUTUM

| Observation | Method | Integration |
|-------------------------|----------------------------|---|
| Elastic tissue | Unstained | Pulmonary excavation lung abscess (p 2214) cavernous tuberculosis (p 2199) |
| Curschmann's spirals | Unstained | Bronchial asthma (p 2101) |
| Charcot Leyden crystals | Unstained | Bronchial asthma (p 2101) |
| Pigmented cells | Unstained | Pneumoconiosis (p 2065) chronic passive congestion (p 2086) |
| Ova | Unstained | Lung fluke (p 2213) |
| Eosinophils | Wright stain | Bronchial asthma (p 2101) |
| Cocci | Gram stain | Gram positive most likely pneumococci (p 199) streptococci (p 157) or staphylococci (p 151) |
| Bacilli | Gram stain | Gram negative most likely influenza (p 396) pertussis (p 278) or Friedländer (p 329) |
| Acid fast bacilli | Carbolfuchsin stain (p 52) | Tubercle bacilli (p 252) |
| Spirochetes | Darkfield | Spirochetal bronchitis (p 329) |
| Fungi | Unstained | Systemic mycoses (p 485) |

TABLE 186A—SPECIAL METHODS FOR EXAMINING SPUTUM

| Method | For |
|--|-----------------------------------|
| Concentration with anti-formin (p 264) | Tubercle bacillus |
| Neufeld typing (p 201) | Pneumococcus |
| Injection into mouse peritoneum (p 63) | Pneumococcus |
| Injection into guinea pig (p 69) | Tubercle bacillus |
| Cough plate (p 280) | Influenza pertussis |
| Culture | Redominant organism and pathogens |

See *Differential Diagnosis of Cough* (p 2050) *Hemoptysis* (p 2058)
Commoner Febrile Intrathoracic Disorders (p 404)

THE GASTRO INTESTINAL CONTENTS

Gastric juice duodenal contents bile and feces are examined in the office laboratory

GASTRIC CONTENTS

The contents of the stomach consist of (1) gastric juice and (2) ingested food substances In addition a reflux of duodenal content through the pylorus may stain the admixture with bile

Gastric Juice—The gastric juice is secreted in response to chemical nervous and mechanical stimuli For the production of appetite juice it is not necessary for food to enter the stomach Sham feeding with expulsion of food through an esophageal fistula the thought or sight of food or the conditioning of the secretory reflex—may effect a similar result Distention of the stomach mechanically influences gastric secretion as do secretagogues present in food

The secretion of gastric juice may be inhibited by nervous influences hence the necessity of providing palatable appetizing foods for the debilitated and convalescent

At the height of digestion the gastric juice normally contains (1) water (2) combined hydrochloric acid (3) free hydrochloric acid (4) pepsin (5) lipase (6) rennin and (7) mineral salts

Hydrochloric acid is formed chiefly by the secretory activity of specialized cells in the fundus of the stomach The acid performs several functions It converts the zymogens into active pepsin and rennin It combines loosely with food protein forming acid metaprotein the first step in protein digestion The portion of hydrochloric acid which is loosely combined with protein is called combined hydrochloric acid The acid which remains in excess after the union with the available protein is termed free hydrochloric acid

The enzymes are secreted by the gastric glands as zymogens (pre enzymes) The activated pepsin in conjunction with the free hydrochloric functions to digest the available protein The rennin produces curdling of milk Lipase a fat splitting enzyme has very little gastric activity

Stomach Contents Other Than Gastric Juice—Ingredients of extragastric origin may be present in the stomach These include food particles bile and products of inflammation such as blood pus mucus or fragments of neoplastic material Where there has been advanced destruction of gastric tissue such organic acids as lactic acid may actually appear in the stomach content as a result of bacterial activity

STANDARD TEST MEALS

Standard test meals are used to promote the secretory activity of the gastric glands in order to obtain material for gastric analysis and to test the functional capacity of the acid secreting cells

The motility meal consists of a handful of raisins eaten the night before the test In the presence of impaired motility or obstruction the

raisins may be detected grossly when the fasting contents of the stomach are removed the following morning

TABLE 187—EXAMINATION OF GASTRIC CONTENTS

| <i>Macroscopic</i> | |
|---|--|
| | Interpretation |
| Amount Fasting content | Quantity in excess of 100 cc suggests pyloric stenosis (p 1780) or hypersecretion (p 1740) |
| Color Bile stained Blood stained Coffee ground | Normal reflux through pylorus Trauma ulceration (p 1762) Bleeding and retention as in carcinoma (p 1814) |
| Mucus Excess amount | Secretory disturbance (p 1740) |
| Odor Fecal | Intestinal obstruction (p 1803) |
| Gross Particles Raisins (after test meal) | Pyloric obstruction (p 1789) |

Chemical and Microscopic

| | Technic | Interpretation |
|---------------------|---------------|--|
| Free HCl | See p 3723 | Normal range 20-60 cc N/10 sodium hydroxide per 100 cc of gastric content See also Hyperacidity (p 1740) subacidity (p 1740) achylia gastrica (p 1768) primary hyperchromic anemia (p 1077) |
| Total Acidity | p 3724 | Normal range 40-80 cc N/10 sodium hydroxide per 100 cc of gastric content |
| Lactic Acid | p 3725 | Gastric achylia and retention as in carcinoma (p 1814) |
| Gastric Ferments | | Absent only in advanced malignancy |
| Blood | p 3728 | Trauma ulceration (p 1762) malignancy (p 1814) |
| Tubercle Bacilli | p 52 | Swallowed sputum |
| Boas Oppler Bacilli | p 3723 | Carcinoma (p 1814) |

* Should be referred to Clinical Pathologist

The *standard test meals* are ingested by the patient following the removal of the fasting contents. They should be sufficiently simple so that they do not interfere with the examination of the gastric juice itself

The classic *Ewald breakfast* consists of a roll and 300 to 400 cc of water. A Shredded Wheat biscuit may be substituted for the roll. For the *alcohol meal* the patient drinks 50 cc of 7 per cent alcohol. The *Rehfsuss test meal* consists of a bowl of gruel. The more complicated meals have no advantage over these simple meals. The presence in them of steak for example invalidates the tests for blood.

ROUTINE EXAMINATION

The routine examination of the gastric content may be completely carried out as follows. At bed time the patient is given a handful of *rainns* to swallow. Thereafter no food drink or drugs are permitted. In the morning the *stomach tube* is passed and the fasting gastric content is removed for examination. The *Rehfsuss* and *Lewine tubes* (p 1750) are best adapted for these purposes.

The test meal is then swallowed or injected through the tube. Specimens of the combined test meal and the gastric juice are withdrawn at intervals. In the absence of free *HCl* the patient receives hypodermically



FIG. 1096.—B as Oppler ba ill from case of gastr an er (Boston)

0.5 mg of *histamine hydrochloride*. Fractional specimens are again withdrawn to determine the effect of the drug on the secretion of acid. *Histamine* is a powerful stimulator of the acid secreting cells of the stomach. The absence of hydrochloric acid in the gastric juice after histamine denotes a *true achlorhydria* (p 1768).

Chemical Examination (Office Laboratory).—*Free and Total acidity*.—The acidity of the gastric contents is measured by titration with tenth normal sodium hydroxide using *Topfer's reagent* (*dimethyl amino azobenzene* in 0.5 per cent alcoholic solution) as the indicator for the *free acid* and 1 per cent alcoholic solution of *phenolphthalein* as the indicator for the *total acid*.

To 2 cc of the filtered gastric juice in an evaporating dish add one to two drops of the indicators. In the presence of *free hydrochloric acid* the *dimethyl amino azobenzene* imparts a *deep cherry red* color to the mixture. With a stirring rod the gastric juice is then vigorously mixed. Tenth normal sodium hydroxide is added drop by drop until the *cherry red* color gives way to *canary yellow*. The amount of alkali employed for this reaction is then noted and gives the reading for free acid.

The titration is continued. When all of the total acid has been neutralized the phenolphthalein indicator will impart a characteristic pink color. A second reading is made of the amount of alkali employed. This figure is used in the calculation of the total and combined hydrochloric acid of

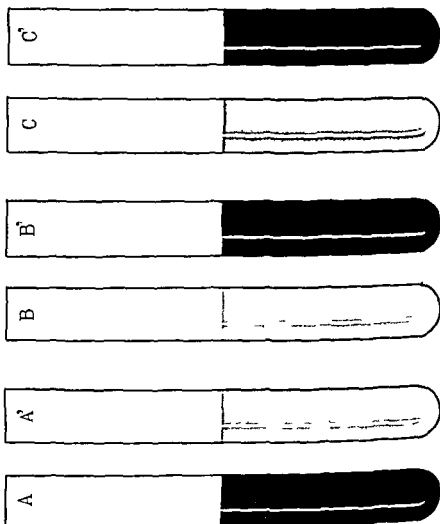


Fig 1097—A Gastric fluid to which a few drops of a 0.5 per cent solution of dimethyl aminoazobenzol has been added. A' After titration with a decinormal solution of sodium hydroxid. B Gastric fluid to which a 1 per cent solution of phenolphthalein has been added. B' B after titration with a decinormal solution of sodium hydroxid. C Gastric fluid to which a 1 per cent solution of alizarin has been added. C' C after titration with a decinormal solution of sodium hydroxid. (Todd and Sanford: Clinical Diagnosis by Laboratory Methods)

the gastric contents. The total hydrochloric acid is represented by the total amount of sodium hydroxide used in both titrations. Combined HCl is calculated from the second titration alone.

Specimens in which there is no free hydrochloric acid will not develop the initial cherry red color from the Topfer's reagent. They all appear

yellow at first changing gradually to pink as the phenolphthalein end point is reached

CALCULATION—The calculation of the free and total acidity is commonly expressed as the number of cubic centimeters of $N/10$ sodium hydroxide per 100 cc of gastric content. Consequently if 2 cc are employed in the test as outlined above the acid figure is multiplied by 50 to obtain the reading

INTERPRETATION—The most important information concerning the acid secretion is the presence or absence of free hydrochloric acid. While free hydrochloric acid is frequently absent from the normal gastric contents a flow should be initiated when the patient receives an injection of histamine hydrochloride. The continued absence of free hydrochloric acid despite exhibition of the drug suggests the possibility of complete atrophy or destruction of the acid cells such as might occur in hyperchromic anemia (p 1077) or carcinoma of the stomach (p 1814)



Fig 1008—General view of the gastric contents a Squamous epithelial cells from esophagus and mouth b leukocytes c cylindrical epithelial cells d muscle fibers e fat droplets and fat crystal f starch granules g chlorophyll-containing vegetable matters h vegetable spirals bacteria k sarcinae l yeast cells (Jakob)

Lactic Acid—If free hydrochloric acid cannot be demonstrated after the use of histamine hydrochloride the gastric content should be examined for lactic acid. The presence of this organic acid has ominous implications. It is most likely due to the presence of a malignant neoplasm which produces achlorhydria and allows lactic acid forming bacteria to grow in the stomach.

TECHNIC OF TEST FOR LACTIC ACID—A gross test for lactic acid is simply performed. The gastric juice is filtered and 3 drops of 10 per cent ferric chloride are added. The presence of lactic acid is indicated by a color change to a canary yellow. As a control an equal amount of water is placed in a second test tube to which is added a similar amount of the ferric chloride. The color may then be compared to the control by looking down on the tops of the test tubes.

Microscopic Examination (Office Laboratory)—Microscopic examination of the gastric contents is performed by placing a drop of unfiltered contents on a slide and examining under the low and high dry objectives. The addition of a drop of Lugol's solution will aid in distinguishing various

structures Under normal conditions little is seen except great numbers of *starch granules* with an occasional *epithelial cell* *yeast cell* or *bacterium* Starch particles stain blue with the iodine solution when undigested and reddish when digested

Pathologically, *remnants of food* from previous meals *erythrocytes* *leucocytes* *yeast cells* and *bacteria* are found Packets of *B. sarcinae* which have a characteristic 'cotton bale' appearance, signify fermentation They rarely occur in cancer of the stomach Huge *Boas* *Oppler bacilli* occur in ulcerating carcinomas with achlorhydria

Tubercle bacilli may be identified in the gastric contents of infected patients who swallow sputum Before aspirating the stomach for this purpose the patient should be put on a milk and butter free diet for three to four days Acid fast bacilli present in these foods may be the source of a false positive result Examination of the gastric content for tubercle bacilli is especially valuable in infants and children with pulmonary tuberculosis

TABLE 188—TYPICAL FINDINGS IN GASTRIC CONTENTS*

| | Normal | Gastric Ulcer | Gastric Cancer | Hyperchromatic |
|----------------------|----------------|---------------|----------------|----------------|
| Odor | Slightly sour | Slightly so | Fil | Colorless |
| Amount | Less than 4 oz | 4-15 oz | 10-25 | 2-6 oz |
| Mucous | Slight | Cloudy | Mucous | Scanty |
| Blood | None | Sometimes | Brown | Rare |
| Free HCl (turbidity) | 5-50 | 30-90 | 0 | 0 |
| After test meal | 70-80 | 50-100 | 0-25 | 0 |
| After histamine | 70-140 | 80-150 | 0-100 | 0 |
| Lactic acid | 0 | 0 | | 0 |

* Holmes

See *Differential Diagnosis of Dyspepsia* (p 1770) *Polyphagia* *Bulimia* and *Pica* (p 1776) *Anorexia* (p 1779) *Epigastric Pain* (p 1788), *Epigastric Swellings and Tumors* (p 1814) *Hematemesis* (p 1764) *Melena* (p 1843)

DUODENAL CONTENTS

The duodenal contents are collected when the *Rohruss tube* (p 1750) passes beyond the pylorus

THE DUODENAL FLUID

Normal duodenal fluid is clear colorless or light yellow depending upon the amount of mixture with bile It is distinctly viscid and slightly alkaline to litmus Under normal circumstances the fluid will drip from the free end of the tube If the fluid is more than usually viscid however it may be necessary to start the drainage by siphonage When approximately 10 to 20 cc of the duodenal fluid have been obtained *biliary drainage* (p 1752) is instituted Meantime the specimen of duodenal contents is kept for examination

BILIARY DRAINAGE

After collection of the duodenal contents 80 cc of magnesium sulfate (25 per cent) is instilled slowly into the duodenal tube The collection of the duodenal contents is continued Under normal circumstances a golden

yellow bile appears shortly. This specimen is designated *A bile* and is thought to be chiefly from the common duct bile.

After a few minutes the *A bile* is replaced by a darker more viscid bile supposedly emanating from the gallbladder. This portion usually amounts to from 30 to 75 cc and is designated *B bile*.

Finally a clear light yellow bile of low specific gravity which is assumed to be freshly secreted bile from the liver appears. This is designated as *C bile*.

Chemical Examination—The duodenal and biliary fluid can also be examined chemically for the presence of *ferments*. This examination is quite complicated and rarely gives important clinical information.

Bacteriologic Examination.—The bacteriologic characteristics of the fluids provided they can be collected under aseptic conditions may be of the utmost significance particularly in the detection of *typhoid carriers*.

TABLE 189.—ANALYSIS OF DUODENAL CONTENTS

| A Bile | B Bile | C Bile | Interpretation |
|---|----------------------------------|--|-----------------------------------|
| Variable amount golden yellow fluid consistency | 30-75 cc dark viscid consistency | Variable amount light yellow fluid consistency | Normal |
| Absent | Absent | Absent | Common Duct Obstruction (p. 2004) |
| Present | Absent | Present | Cystic Duct Obstruction (p. 1992) |
| Clear | Pus | Clear | Cholecystitis (p. 2009) |
| Cloudy | Cholesterol | Clear | Cholelithiasis (p. 1997) |
| Clear | Clear | Pus | Cholangitis (p. 2010) |
| Cloudy | Bacilli | Bacilli | Cholecystitis and cholangitis |
| Blood | Blood | Blood | Malignancy (p. 199) |

EXAMINATION OF THE STOOL (ROUTINE)

Routine examination of the stool is usually neglected in practice because of the unpleasantness of collecting and examining feces. The specimen should be passed in a clean chamber. The patient should observe the approximate size and thickness of the entire specimen and note its consistency, odor, and the presence of gas. With the aid of a wooden tongue blade a small fragment is picked out and placed in an ointment jar.

A minimum examination consists of a description of the (1) *physical properties* (shape, consistency, color, and odor), (2) the test for occult blood, (3) *microscopic examination*.

The Normal Stool—The normal stool is a mixture of (1) *water*, (2) *undigested and indigestible remnants of food*, (3) *digested foods* excreted before absorption can take place, (4) *products of the digestive tract* (altered bile pigments, enzymes, and mucus), (5) *products of decomposition*.

tion (skatol indol fatty acids and various gases), (6) epithelial cells shed from the wall of the intestinal canal, (7) bacteria which constitute about one third of the weight of the dry stool

See *Differential Diagnosis of Diarrhea* (p 1840) *Melena and Tarry Stools* (p 1843) *Constipation* (p 1852) *Pruritus Ani* (p 1916) *Ano-*

TABLE 190 — EXAMINATION OF STOOL

| Macroscopic | |
|--|---|
| | Interpretation |
| Gross appearance Moulded and dry Cigarette shape with twirled ends Ribbon shaped Foamy | Rectal constipation Spastic constipation (p 1846) Obstruction annular carcinoma (p 1888) Sprue pancreatic insufficiency (p 1938) |
| Odor Acrid Putrid Sulfur | Bacterial decomposition excess fat Protein decomposition Cathartic |
| Color Light yellow Dark brown Dark green Red Black Clay Burgundy red Tarry | Milk diet rhubarb senna santonin unchanged bilirubin Chocolate berries cherries Spinach calomel biliverdin Beets or blood Fruit charcoal iron bismuth blood Obstructive jaundice excessive fat Excessive and massive bleeding from lower intestinal tract Bleeding from upper intestinal tract |
| Mucus | Inflammation mucous colitis (p 1846) |

Chemical and Microscopic

| | Technic | Interpretation |
|-------------------|---------|--|
| | See | |
| Occult Blood | p 3728 | If on meat free diet bleeding has occurred |
| Bile Pigments | p 3728 | Absent in obstructive jaundice |
| Ova and Parasites | p 3732 | Intestinal infestation |
| Amebae | p 3730 | <i>E coli</i> or <i>E histolytica</i> |
| Flora | p 372 | Gram positive Gram negative |
| Tubercle Bacilli | p 3690 | From swallowed sputum especially in children |

perineal Pain (p 1913) *Incontinence of Feces* (p 1915) *Diarrheas in Infancy* (p 2782)

Chemical Examination (Office Laboratory)—*Occult Blood*—The stool should be examined for the presence of occult blood. A few benzidine crystals are placed in a test tube containing a few drops of glacial acetic acid. To this suspension are added about 5 cc of a thin fecal suspension

that has been previously boiled and cooled. A few drops of hydrogen peroxide are then placed in the tube. In the presence of blood an intense blue color develops within a few moments. If the benzidine reaction is positive the patient should be examined for obvious local bleeding as from hemorrhoids or bleeding gums. To exclude error additional specimens are examined after three days on a meat free diet. If the test is still positive it signifies bleeding into the gastro intestinal tract. See *Differential Diagnosis of Melena and Tarry Stools* (p 1843).

Starch and Fatty Acid—The fecal suspension is examined for the presence of excessive starch and fatty acid. With the addition of a drop of tincture of iodine an intense blue color develops in the presence of undigested starch. To another small fraction of the suspension a drop of Sudan III stain is added. An intense pink color indicates the presence of excessive neutral fat.

Motility Test—Ordinarily adults on a mixed diet require twenty to thirty six hours for the passage of ingested material with infants the time is less. The time of passage is ascertained by giving 0.5 gm. of powdered charcoal or 0.3 gm. of carmine in a capsule and noting the appearance of the color in the stool. See *Differential Diagnosis of Constipation* (p 1852) *Diarrhea* (p 1840).

Bile Pigments in the Stool—The presence of bile pigment in the stool is evidenced grossly by the color. A clay colored or whitish stool suggests the absence of bile pigment but such a stool may also be seen after a barium enema or a barium meal. A simple qualitative test for bile pigment may be performed by the method of Schmidt. A small quantity of fecal matter is placed in a porcelain dish rubbed together with saturated mercuric chloride solution and allowed to stand for twenty four hours. A red color at the end of that time indicates the presence of urobilin. A green color shows the presence of bilirubin which is not seen normally.

A more rapid test is done by filtering 10 to 15 cc. of a moderate fecal suspension. The filter paper is allowed to dry for fifteen to twenty minutes and is placed in a porcelain dish. A drop of concentrated nitric acid is placed on the paper in an area where the feces have passed through. A series of colored rings (red orange green) denotes the presence of bile. See *Differential Diagnosis of Obstructive Jaundice* (p 1954).

Microscopic Examination—The routine stool survey is concluded with a microscopic examination for ova and parasites. Most of these are easily recognized in the normally passed specimen which need not be freshly examined except in the instance of a search for the mobile ameboid stage of the *Endamoeba histolytica*. For the detection of the latter catharsis may be required and immediate microscopy is mandatory for recognition of the organisms.

Contrary to the general opinion the undernoted axioms may be accepted on the basis of extensive experience in the Army and Navy. (1) Catharsis is not needed except in the search for the *Endamoeba histolytica*. (2) Other than in the search for the same organism stools need not be freshly examined but may be kept in the ice box until an opportune moment. (3) A single examination will reveal 75 per cent of infections and a series of three stools should disclose all of the infecting organisms. (4) Concentration methods are rarely required.

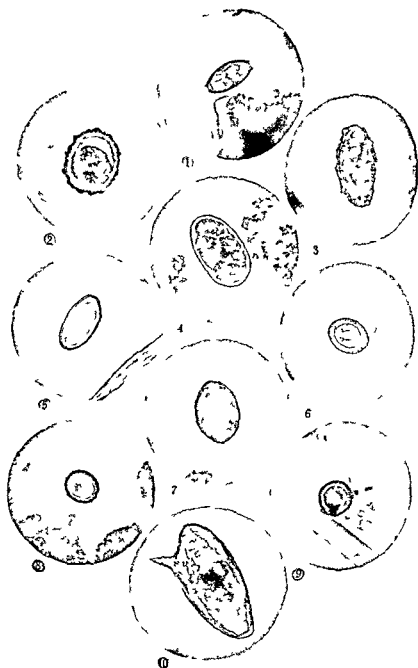


Fig 1099.—Ova which may be found in the feces showing comparative size (photographs $\times 200$) 1 *Trichuris trichiura* (whipworm) 2 *Ascaris lumbricoides* (roundworm) fertilized 3 *Ascaris lumbricoides* unfertilized 4 *Necator americanus* (hookworm) four-cell stage 5 *Enterobius vermicularis* (pinworm) 6 *Hymenolepis nana* (dwarf tapeworm) 7 *Diphylllobothrium latum* (fish tapeworm) the edge of the lid being out of focus 8 *Taenia saginata* (beef tapeworm) 9 *Taenia solium* (pork tapeworm) lying beside a striated muscle fiber 10 *Schistosoma mansoni* (blood fluke) *

For routine microscopic work the wet smear is examined in normal saline solution in water and after staining with iodine.

The Wet Smear in Normal Saline—A small particle of feces is selected with a toothpick and stirred in a drop of normal saline on a clean slide. When a smooth emulsion is formed a cover slip is placed perpendicularly in the emulsion and moved along the slide for about an inch carrying the preparation along. By this technic air bubbles are excluded from the surface when the cover slip is lowered. The film should be sufficiently thin so that newsprint can be read through it.

The saline smear reveals helminth ova and larvae and the trophozoites (motile adult forms) and cysts of intestinal protozoa. Ova and larvae are easily noted with the low power of the microscope; trophozoites and cysts appear as flashing blue points of light under low power and require greater magnification for identification. The low power objective is most



Fig. 1100.—Early exudate in stool; young and old trophozoites of *E. histolytica* in stool; scanty exudate.

valuable for the preliminary recognition of intestinal protozoa since the characteristic blue points of light stand out clearly in a properly made smear.

The Wet Smear in Water (Aqueous Smear)—A thin smear with tap water is a valuable aid in the recognition of protozoan cysts since it causes trophozoites and blastocysts (plant cells) which resemble amebic cysts to disintegrate.

The Wet Smear Stained with Iodine—A third smear is made with water and lightly tinted with a small drop of Lugol's solution. The iodine quickly kills trophozoites and cysts of intestinal protozoa and visualizes the nuclei and glycogen bodies. It is of value in the diagnosis of endamoeba infections since the number and arrangement of the nuclei may be detected. Too much iodine lessens the contrast between the nuclei and cytoplasm and makes recognition more difficult.

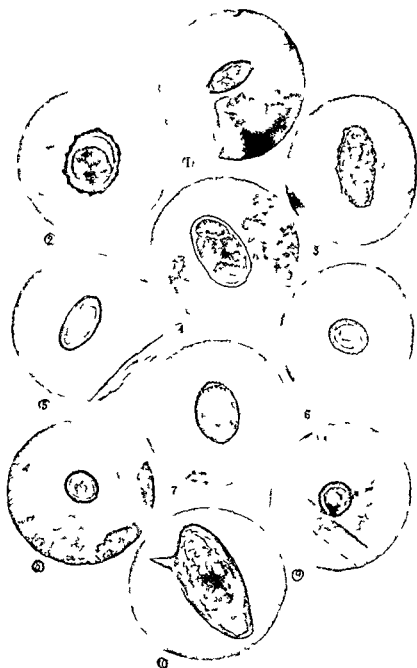


Fig 1099.—Ova which may be found in the feces showing comparative size (photographs $\times 250$) 1 *Trichuris trichiura* (whipworm) 2 *Ascaris lumbricoides* (roundworm) fertilized 3 *Ascaris lumbricoides* unfertilized 4 *Necator americanus* (hookworm) four-cell stage 5 *Enterobius vermicularis* (pinworm) 6 *Hymenolepis nana* (dwarf tapeworm) 7 *Diphyllobothrium latum* (fish tapeworm) the edge of the lid being out of focus 8 *Taenia saginata* (beef tapeworm) 9 *Taenia solium* (pork tapeworm) lying beside a striated muscle fiber 10 *Schistosoma mansoni* (blood fluke) *

TABLE 192.—DIFFERENTIAL CHARACTERISTICS OF INTESTINAL PROTOZOA (TROPHOZOITES) AS SEEN IN NORMAL SALINE AND AQUEOUS SMEARS

| <i>Trophozoites (L. may Ameboid Form)</i> | | | | | |
|---|-------------------------------------|----------------------------|--|---|----------------------------|
| | Size: Microns | Shape at Rest | Motility | Cytoplasm | Visibility of Nucleus |
| <i>E. histolytica</i> | 8 to 40 Small race 8. Large race 18 | Slightly irregular circle. | Tongue-like pseudopod protrude suddenly progress crawl in one direction & agglutinate. | Clean cent no ingested RBC in amebiasis. | Usually not seen. |
| <i>E. coli</i> | 20 (10 to 50) | Same as above | Crawls & drag in h. Crawl in one direction rare. | Dirty due to detritus & bacteria. Ingested RBC. rare. | Visible in all forms. |
| <i>E. nana</i> | 8 (6 to 14) | Same as above | Small, knob-like pseudopods, no progression. | Same as <i>E. coli</i> . | Rarely visible. |
| <i>I. butschlii</i> | 10 (6 to 40) | Same as above | Same as <i>E. coli</i> . | Same as <i>E. coli</i> . | Same as <i>E. nana</i> . |
| <i>O. fragilis</i> | 12 (6 to 18) | Perfect circle. | Thin veil-like, triangular or rectangular pseudopodia with hairy points. | Same as <i>E. coli</i> . | Not visible. |
| <i>T. hominis</i> | 8 to 10 | Ovoid | Nervous, rapid, jerky | Flagella and undulating membrane. | Rarely visible. |
| <i>C. mesnili</i> | 15 (14 to 18) | Cone-shaped | Rolling slow progression, slow rotation of body | Spiral groove. | Rarely visible. |
| <i>G. lamblia</i> | 15 (14 to 15) | Pear-shaped | Slow tumbling, leaf-like. | Parallel axostyles. | Visible. |
| <i>B. coli</i> | 50 X 70 | Oval | Dark across field body rotates slowly | Cytostome contractile vacuoles. | Free living rows of cells. |

TABLE 193.—DIFFERENTIAL CHARACTERISTICS OF INTESTINAL PROTOZOA (CYSTS) AS SEEN IN NORMAL SALINE AND AQUEOUS SMEARS

| <i>Cysts</i> | | | | | |
|-----------------------|-------------------------------------|--------------------------|---|--|------------------------------------|
| | Size in Microns | Shape at Rest | Chromatoid Bodies | Glycogen | Visibility of Nucleus |
| <i>E. histolytica</i> | 8 to 16 Small race 8. Large race 12 | Round or oval. | Early seen bars with rounded ends. Present in 80%. | Rarely floats appearance of cyst. | Visible only in old cysts. |
| <i>E. coli</i> | 17 (10 to 50) | Same. | Scanty, blocks and spindles with pointed ends. Seen in 10 per cent. | Fills cytoplasm of young cysts. | Visible. More than 4 distinct. |
| <i>E. nana</i> | 8 (6 to 10) | Round, oval or football. | None | Not visible. | Not visible. |
| <i>I. butschlii</i> | 10 (6 to 16) | Pear or kidney shaped. | None | Well defined large refractile glycogen ball. | Rarely visible. |
| <i>C. mesnili</i> | 8 | Lemon with narrow tip. | None | None | None trace of nucleus or flagella. |
| <i>G. lamblia</i> | 8 X 12 | Football | None. Filicils, rods and rings. | In young cysts. | Visible. |
| <i>B. coli</i> | 50 X 80 (rare) | Oval | None | | Kidney-shaped macronucleus. |

TABLE 191—MORPHOLOGICAL FEATURES OF HELMINTH OVA FOUND IN FECES

| | Size (Average Microns) | Morphology |
|--|---------------------------|---|
| <i>Schistosoma mansoni</i> (blood fluke) | 155 × 60 | Light yellow semitransparent oval structure with a lateral spine pointing backward |
| <i>Schistosoma haematobium</i> | 148 × 60 | Light yellow semitransparent elongated oval with a terminal spine |
| <i>Schistosoma japonicum</i> | 80 × 67 | Light yellow almost circular structure without a spine (or a rudimentary one) |
| <i>Ascaris lumbricoides</i> (roundworm) | 90 × 38 (unfertilized) | Roughly globular yellowish brown structure with an irregular outline Considerable variation in shape |
| <i>Ascaris lumbricoides</i> (roundworm) | 58 × 42 (fertilized) | Yellowish brown thick shelled oval structure with or without an irregular rough albuminous coating Contains unsegmented cytoplasm separated at each pole from shell by a clear space (crescentic) |
| <i>Diphyllobothrium latum</i> (fish tapeworm) | 70 × 45 | A yellowish brown oval structure with a thin wall filled with small spheres and provided with a hinged operculum at one end |
| <i>Ancylostoma</i> or <i>Necator</i> (hookworm) | 58 × 37 | Oval colorless transparent structure with a thin smooth shell Embryo divided into 2, 4 or 8 segment |
| <i>Enterobius vermicularis</i> (pinworm) | 55 × 26 | Oval colorless thin shelled structure flattened on one end and containing partially developed coiled embryo Rare in feces Present on anal skin |
| <i>Trichuris trichiura</i> (whipworm) | 52 × 22 | Brown thick shelled barrel shaped egg with colorless terminal plugs |
| <i>Hymenolepis nana</i> (dwarf tapeworm) | 47 × 37 | Colorless semitransparent oval structure containing an embryo separated from the outer shell by a broad gelatinous zone At each pole of embryo are 2 distinct filamentous processes Three pairs of hooklets may be seen in embryo |
| <i>Taenia saginata</i> (beef tapeworm) <i>Taenia solium</i> (pork tapeworm) | 35 | Brown spherical structures with a thick radially striate shell Embryo contains six hooklets seen as three pairs of parallel lines |
| <i>Clonorchis sinensis</i> (fluke) | 29 × 16 | Brown structure shaped like an old fashioned light bulb Has an operculum at narrow end and a knob on round end |

* Ova arranged according to size in microns (red blood cell diameter equals 7.5 microns)

Permanent Smear—The permanent iron hematoxylin smear is referred to the clinical pathologist for confirmation of the presence of *E. histolytica* cysts

infants the normal pressure in the recumbent position is 50 to 100 mm. Removal of 10 cc of fluid normally causes a drop of about 50 mm in pressure.

Elevated initial pressure indicates increased intracranial pressure (p 1431) transmitted freely to the spinal subarachnoid space.

When the normal reading has been established pressure is applied first to the right jugular vein and then the left. In normal individuals this should produce a marked rapid

TABLE 194—EXAMINATION OF CEREBROSPINAL FLUID

| | Technic | Interpretation |
|------------------------------|------------|---|
| Increased Initial Pressure | See p 3435 | Increased intracranial pressure (p 1431) (over 200 mm H ₂ O) |
| Decreased Initial Pressure | p 3735 | Partial subarachnoid block (p 1434) (less than 100 mm H ₂ O) |
| Negative Queckenstedt Test | p 3537 | Subarachnoid block (p 1434) |
| Bloody Fluid | p 1445 | Trauma subarachnoid hemorrhage (p 1445) |
| Opalescent Fluid | p 214 | Infection meningitis (p 214) |
| Yellow Fluid (Xanthochromia) | p 1445 | Old blood subarachnoid hemorrhage (p 1445) spinal cord compression (from synovium p 1430) |
| Congulum on Standing | p 202 | Tuberculous meningitis (p 202) |
| Lymphocytosis | p 3698 | Nonsuppurative meningitis or encephalitis (p 442) |
| Leukocytosis | p 3698 | Purulent meningitis meningococcus (p 214) |
| Gram negative cocci | p 157 | Meningococcus meningitis (p 214) |
| Gram positive cocci | p 137 | Streptococci pneumococci rhinogenic (p 2128) and otogenic meningitis (p 2148) |
| Globulin | p 3736 | Increase in acute inflammations tuberculous and syphilitic meningitis cord and brain tumors and subarachnoid hemorrhage |
| Sugar | p 3674 | Absent in pyogenic and tuberculous meningitis |
| Serologic Tests | p 336 | Cerebrospinal syphilis (p 1377) |
| Colloidal Gold Curve | p 677 | Paresis (5-5554 211) Tabes (0123443211) Meningitis (0112345320) |

Should be referred to Clinical Pathologist.

rise of pressure in the manometer tube with a corresponding rapid drop back to normal on release. This is known as the Queckenstedt test. In the presence of block somewhere along the subarachnoid space pressure on the right, left or both jugulars may result in a slow and/or diminished rise in pressure and a delayed fall on release. Block prevents the immediate transmission of the artificially elevated venous pressure (i.e. by jugular compression) to the spinal fluid. Normally the rise and fall is marked and rapid. Failure of jugular

CHAPTER 169

LABORATORY METHODS OF CEREBROSPINAL FLUID, TRANSUDATES AND EXUDATES, BASAL METABOLIC RATE, VITAL CAPACITY, DIAGNOSTIC ROENTGENOLOGY

CEREBROSPINAL FLUID

THE cerebrospinal fluid filling the cavities of the brain and surrounding the brain and spinal cord originates from the choroid plexus in the lateral and fourth ventricles. The fluid is formed by the filtration of blood plasma through the semipermeable capillary endothelium of the choroid vessels. The colloids are held back but salts and organic substances of small molecular weight are allowed to pass through.

The pressure of the fluid is about equal to the intracranial venous pressure but is only one sixth of the arterial pressure. The flow of the fluid from the blood to the cerebrospinal system may be reversed by an increase in the osmotic pressure of the blood (as in dehydration or after the injection of hypertonic salt or sugar solutions). With inflammation of the meninges the capillary cells in the meningeal vessels become more permeable and allow protein molecules to pass into the cerebrospinal fluid from the blood.

CIRCULATION

An adult has 100 to 150 cc of cerebrospinal fluid which circulates from within the cavities of the brain to its surfaces. The normal fluid is clear and colorless.

The ventricles are connected by foramina. The fluid escapes from the roof of the fourth ventricle into the *cisterna magna* which is below and behind the brain. Here the course of the fluid divides: four fifths going up over the brain and one fifth down around the spinal cord. The fluid which surrounds the brain flows anteriorly beneath the *medulla pons* and *midbrain*. From here it passes up along the internal carotids and spreads in the sulci which cover the brain surface. The smaller portion of fluid surrounds the spinal cord. There is a small upward flow in the *central canal* of the cord itself. Blockage of the fluid exit from the ventricles causes internal *hydrocephalus* (p 1409).

ABSORPTION

Absorption of cerebrospinal fluid takes place by osmosis through the *arachnoid villi* which are diverticula projecting into the longitudinal and other large sinuses. These villi hypertrophy in adult life and are called *pachymen bodies*. Absorption also occurs by osmosis into the blood capillaries in the cerebral sulci.

In the spinal canal some fluid is absorbed into the blood vessels in the subarachnoid space and the lymph spaces of the lumbar region.

HYDRODYNAMICS

Cerebrospinal fluid for examination is usually obtained by *lumbar puncture* (p 381) commonly performed at the level of third or fourth lumbar vertebra. It may also be obtained by (1) *cisternal puncture* (p 3783) or (2) *ventricular puncture* (p 383). After the needle has entered the subarachnoid space and spinal fluid appears a calibrated tube (*Ayer manometer*) is attached for estimation of hydrostatic pressure.

The normal pressure for the adult in the horizontal position is 100 to 200 mm of water. With the patient upright, the normal reading is doubled i.e. 200 to 400 mm. In

infants the normal pressure in the recumbent position is 50 to 100 mm. Removal of 10 cc of fluid normally causes a drop of about 50 mm in pressure.

Elevated initial pressure indicates increased intracranial pressure (p 1421) transmitted freely to the spinal subarachnoid space.

When the normal reading has been established pressure is applied first to the right jugular vein and then the left. In normal individuals this should produce a marked rapid

TABLE 194—EXAMINATION OF CEREBROSPINAL FLUID

| | Technic | Interpretation |
|------------------------------|------------|---|
| Increased Initial Pressure | See p 3735 | Increased intracranial pressure (p 1421) (over 200 mm H ₂ O) |
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| Negative Queckenstedt Test | p 3737 | Subarachnoid block (p 1434) |
| Bloody Fluid | p 1445 | Trauma subarachnoid hemorrhage (p 1445) |
| Opaque Fluid | p 214 | Infection meningitis (p 214) |
| Yellow Fluid (Xanthochromia) | p 1445 | Old blood subarachnoid hemorrhage (p 1445) spinal cord compression (From syndrome p 1430) |
| Coagulum on standing | p 202 | Tuberculous meningitis (p 202) |
| Lymphocytosis | p 3698 | Nonsuppurative meningitis or encephalitis (p 442) |
| Leukocytosis | p 3698 | Purulent meningitis meningococcus (p 214) |
| Gram negative cocci | p 137 | Meningococcus meningitis (p 214) |
| Gram positive cocci | p 137 | Streptococcus pneumococcus, rhinogenic (p 2128) and otogenic meningitis (p 2148) |
| Clotting | p 3736 | Increase in acute inflammations tuberculous and syphilitic meningitis cord and brain tumors and subarachnoid hemorrhage |
| Sugar | p 3674 | Absent in pyogenic and tuberculous meningitis |
| Serologic Tests | p 336 | Cerebrospinal syphilis (p 1377) |
| Colloidal Gold Curve | p 8737 | Paresis (5555543211) Tabes (0123453211) Meningitis (0112345320) |

Should be referred to Clinical Pathologist

rise of pressure in the manometer tube with a corresponding rapid drop back to normal on release. This is known as the *Queckenstedt test*. In the presence of block somewhere along the subarachnoid space pressure on the right, left or both jugulars may result in a slow and/or diminished rise in pressure and a delayed fall on release. Block prevents the immediate transmission of the artificially elevated venous pressure (i.e. by jugular compression) to the spinal fluid. Normally the rise and fall is marked and rapid. Failure of jugular

compression to raise the pressure may be due also to a partial or complete thrombosis of the jugular bulb on the side to which pressure is applied

Normally there is a rapid rise in the spinal fluid pressure on abdominal compression and straining. A low pressure may be due to a partial or complete block in the spinal subarachnoid space

COLLECTION

After the pressure readings have been completed cerebrospinal fluid is collected for examination. The first few drops are discarded since they may contain blood resulting from the trauma of the puncture.

Subsequent specimens are caught in the second, third and fourth test tubes. Normal fluid is clear and colorless.

CHEMICAL EXAMINATION (OFFICE LABORATORY)

The cerebrospinal fluid is examined routinely for the presence of protein and sugar.

Total Protein—The tests for protein are both qualitative and quantitative. The presence of an excess of protein is detected qualitatively by testing for globulin by the method of Pandey.

Globulin—For the globulin test Pandey's solution is prepared by adding 10 gm of phenol to 100 cc of water. This solution is kept in an incubator and shaken frequently.

To perform the test 1 cc of the reagent is placed in a test tube and 1 drop of the cerebrospinal fluid is added. A bluish white cloud indicates the presence of an abnormal amount of globulin. Normally, at least 4 drops of fluid can be added without causing any turbidity of the Pandey's solution.

Sugar—For the sugar reaction Benedict's solution is diluted with equal parts of water. Eight drops of the cerebrospinal fluid are added to 5 cc of the diluted boiled Benedict's solution. The mixture is boiled again and compared as in the test for *glycosuria* (p 3674).

Normal cerebrospinal fluid gives a positive reduction by this method. The fluid normally contains sugar in an amount equal to 50 per cent of the blood sugar level.

Sulfonamides—Sulfonamide levels are determined by methods similar to those used for blood (p 3717). The determinations are of use in regulating the dosage in cases of meningitis.

SEROLOGIC EXAMINATION (CLINICAL PATHOLOGIST)

Routine serologic examination must be made on all clear spinal fluids. The *Wassermann* (p 337), the *Kahn*, the *Mazzoni* and the *Kline* tests are performed exactly as on blood serum. In syphilis of the nervous system and parasyphilis the blood Wassermann is frequently negative when the spinal fluid findings are strongly positive. The spinal Wassermann is rarely negative in the presence of syphilis of the nervous system.

The *Wassermann*, *Kahn* and *Kline* spinal fluid tests should be supplemented by the *colloidal gold reaction* (Lane). In this test various dilutions of the fluid are mixed with a colloidal solution of gold. Under normal conditions the cerebrospinal fluid causes no precipitation and hence no change in color. Abnormal fluids produce precipitation and consequent changes in the color varying with the dilution from red to purple deep blue to pale blue or colorless.

The report of the colloidal gold test is based upon the color changes of the different dilutions. The brilliant red orange is read at 0 red blue at 1 lilac or purple at 2 blue at 3 pale blue at 4 and complete decolorization at 5. The dilutions used are $\frac{1}{10}$ $\frac{1}{20}$ $\frac{1}{40}$ $\frac{1}{80}$ $\frac{1}{160}$ $\frac{1}{320}$ $\frac{1}{640}$ $\frac{1}{1280}$ $\frac{1}{2560}$ $\frac{1}{5120}$.

The report contains ten figures corresponding to the color in each of the various dilutions. The normal result is ten zeros. In *paresis* (p 1377) the first four or five tubes are completely decolorized (5555543211). In *taber* (p 1464) the readings are lower in the early tubes and rise in the third fourth or fifth tubes to fall again in the higher dilutions (0123443211). In *meningitis* the height of the decolorization does not occur until the very high dilution and a typical curve might be 0112345300.

While the test itself is simple to perform the solutions are difficult to prepare and keep. The test should always be done by an experienced serologist.

| | | Dilutions of Serum Fluid with 0.4% NaCl | | | | | | | | | Controls | |
|-------------------------|---|---|------|------|------|-------|-------|-------|--------|--------|----------|---------|
| | | 1:10 | 1:20 | 1:40 | 1:80 | 1:160 | 1:320 | 1:640 | 1:1280 | 1:2560 | 1:5120 | 1:10240 |
| Complete Decolorization | 5 | 0 | 0 | 0 | 0 | 2 | | | 0 | | | 0 |
| Pale Blue | 4 | | | | | | 0 | 0 | | | | |
| Blue | 3 | | | | 0 | 0 | | | 0 | | | |
| Lilac or Purple | 2 | | | 0 | | 0 | 0 | 0 | | | | |
| Red-Blue | 1 | | 0 | | 0 | | | | 0 | 0 | | |
| Brilliant Red-Orange | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |

Fig 1101—Types of reactions in colloidal gold test. 1 Normal cerebrospinal fluid, no reaction. 2 paresis type. 3 tabetic or tabetic type. 4 meningitic type.*

As a substitute for the colloidal gold test some laboratories perform a similar reaction with a solution of gum mastic. The technic and results are quite similar.

BACTERIOLOGIC EXAMINATION (CLINICAL PATHOLOGIST)

Turbid fluid is allowed to sediment and smears are stained with Gram and Wright's stain. If a fibrin clot forms in a turbid or ground glass fluid the coagulum is lifted out and stained for acid fast bacilli. Gram negative diplococci are most likely meningococci while gram positive cocci are streptococci, pneumococci or staphylococci. In a child the finding of a gram negative pleomorphic bacillus suggests the presence of *Hemophilus influenzae B*.

At the time of the collection of the fluid a chocolate agar and a blood agar slant are inoculated directly from the puncture needle using 0.5 cc of fluid. The media are warmed before use and are sent to a bacteriologist without delay. If purulent meningitis is suspected a blood culture is taken as soon as possible.

Direct precipitin tests on the cerebrospinal fluid are of great value in early specific diagnosis. One tenth of a cc. of polyvalent antimeningococcus serum, anti-influenza B serum or polyvalent antipneumococcus serum is placed in a precipitation tube and 0.1 cc. of cerebrospinal fluid is layered over it. A white ring is sought at the point of contact of the fluids and the observation is repeated in an hour after incubation at 37° C. The tube is then shaken and a precipitate is sought at the end of another hour and again after the tube has been in the refrigerator over night.

TABLE 19. — CHARACTERISTICS OF TRANSUDATES AND EXUDATES

| | Transudates | Exudates |
|------------------------|---|---|
| Specific Gravity | Less than 1.018 | Over 1.018 |
| Albumin | Less than 2.5 per cent | Over 2.5 per cent |
| Coagulation | Not observed | Forms clot |
| Histology | Mononuclear cells | Leukocytes |
| Gram Stain | No organisms | Organisms may be demonstrable |
| Bile | Not present | Present in obstructive jaundice (p. 1931) |
| Lymphocytes | Not present | Suggest tuberculosis (p. 252) |
| Leukocytes | Not present | Indicate suppuration |
| Culture | Sterile | May be positive for tuberculosis (p. 252) or other organism |
| Guinea pig inoculation | Examine animal after 3 to 6 weeks for evidences of tuberculosis | Examine animal after 3 to 6 weeks for evidences of tuberculosis |
| Centrifugation | No formed elements | May demonstrate atypical cells with mitotic figures (neoplasm) |
| Fat Globules | Not present | Suggest obstruction of thoracic duct |

If gram positive cocci are found on smears a direct typing with anti-pneumococcus serums is attempted by the Neufeld method (p. 202).

TRANSUDATES AND EXUDATES

Transudates and exudates may be obtained from pleural, peritoneal and joint cavities. Their characteristics are set forth in Table 19.

See *Differential Diagnosis of Ascites* (p. 1921) *Pleural Effusion* (p. 2032)

DETERMINATION OF THE BASAL METABOLIC RATE

The determination of the basal metabolic rate requires the purchase of a *spirometer* and an *oxygen tank*. The test is performed by calculating

the consumption of oxygen with reference to the surface area, sex and age of the patient with corrections for the temperature and barometric pressure.

The manufacturer supplies directions, tables, soda lime, nose clips and mouthpieces.

Spirometers—In a general way there are two types of spirometer. The one calculates the time necessary for the consumption of a given amount of oxygen; the other the amount of oxygen consumed in a given time. Each method is reliable and accurate. The machines are sturdy, require little care and will give complete satisfaction.

Technic.—Though the test is very simple, accuracy demands scrupulous attention to minute detail. The patient retires early the night before the test and arrives at the office in a leisurely manner without having taken food or drink. He rests on a couch from ten to thirty minutes. The clothes should be loosened. Exceedingly restless patients can stand only a short period of relaxation. The longer rest should be employed for the more lethargic individuals, many of whom will fall asleep. The operator explains to the patient that the test should cause no discomfort, that its purpose is to measure the oxygen consumption at rest and that quiet, natural breathing is desirable.

Oxygen is introduced into the spirometer. The system is closed except for the communication between machine and mouthpiece. The nose clip is applied. When normal respiration has been established the test is started. Absolute silence should prevail during the test and the operator should not leave the patient alone. The resting pulse is taken at the beginning and at the end of the test. The respiratory record on the tracing is watched. Any unevenness due to restlessness, swallowing of air or a possible leak necessitates a fresh start. When the operator is satisfied that a really basal reading has been obtained the test is terminated. If the patient becomes progressively uncooperative the test is repeated on another day.

Calculations.—The calculation varies with the type of machine employed. Tables are furnished by the manufacturer. In every instance, however, it is necessary to get the patient's height, weight, sex and age as well as the temperature of the room and the approximate barometric pressure.

Errors.—The possible errors in the determination of the basal metabolic rate follow: (1) Leakage around the mouth or nose pieces or in the machine will give misleadingly high levels of oxygen consumption. (2) Saturation of the soda lime, which removes CO₂, will cause dyspnea. The ordinary supply of soda lime will last for two or three months. (3) Restlessness, anxiety, air swallowing, cough and fever influence readings and produce inaccuracy on the high side.

See *Differential Diagnosis of Increased Basal Metabolic Rate* (p. 720) *Decreased Metabolic Rate* (p. 719).

DETERMINATION OF VITAL CAPACITY

Vital capacity may be measured by the machines used for estimating the basal metabolic rate. The bell of the machine is emptied of oxygen

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Transudates and exudates may be obtained from pleural, peritoneal and joint cavities. Their characteristics are set forth in Table 195.

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DETERMINATION OF THE BASAL METABOLIC RATE

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- 7 A foot switch for fluoroscopy
- 8 A fluoroscopic screen
- 9 Cassettes and films

Technic of Fluoroscopy—With the portable apparatus the following operations are employed for fluoroscopy which may be performed in the office or the home without the necessity of darkening the room or making any other preparations

- 1 Mount the x ray tube head on the tube stand
- 2 Attach the control unit to the outlet for house current. The machine operates only on AC. *It must not be used with direct current.*

TABLE 196 —DIAGNOSTIC ROENTGENOLOGY

| | Positioning | Note Especially |
|-------------------------|--|---|
| Chest Fluoroscopy | Rotate during observation | Lung fields cardiac contours (p 795) great vessels (p 796) lighting of apices movements of diaphragm |
| Bones and Joints | At least two views compare two sides | Fractures (p 2982) dislocations (p 2984) abnormal calcification and decalcification |
| Teleoroentgenograms | 60-72 inch distance from cassette to tube also lateral oblique | Lung fields size of heart measurements of great vessels |
| Scout Films of Abdomen | Anteroposterior and postero-anterior positions | Stones in urinary tract (p 2311) or biliary passages (p 1997) calcified lymph nodes (p 1887) intestinal obstruction (p 1874) pregnancy (p 2620) |
| Skull | Anteroposterior and lateral. | Fracture (p 1450) increased intracranial pressure (p 1468) destruction of sella turcica (p 1177) |
| Nasal Accessory Sinuses | Refer to roentgenologist | Diminished aeration and radiability of involved sinus (p 2126) |

- 3 Attach the foot switch to the control unit
- 4 Turn on the switch of the control unit. The bull's eye light should go on and the voltmeter records the house current
- 5 Set the milliamperage at 5 with the filament regulator
- 6 Place the patient or the part to be fluoroscoped about 18 to 24 inches away from the unit with the compression cone aimed at the site that is to be visualized
- 7 Adjust the fluoroscopic screen to the eyes and visualize the area illuminated by the ray passing from the cone to the examiner's field of vision

Technic of Radiography—To make a radiograph the foot switch is removed and the hand timer is attached to the control unit. The settings

The patient after a deep breath closes his nostrils and through the mouth piece blows the spirometer up to the limit of his capacity. At least three readings are made. A simpler and satisfactory calculation is approximated by multiplying the height in centimeters by 25 for men, by 20 for women and by 29 for athletes. In children the normal is 15 cc for each centimeter of height. The variation from the normal is calculated by the formula $\frac{\text{Reading}}{\text{Normal}} \times 100 = \% \text{ of Vital Capacity}$. Figures in excess of 100 per cent are expressed as plus readings; those below 100 per cent are subtracted from 100 and expressed as minus readings. Thus 125 is plus 25 per cent while 75 is minus 25 per cent.

Interpretation—Several factors other than heart failure influence the vital capacity. Most important is the physiological decrease with *advancing years*. Many types of pulmonary disease diminish the aeration capacity of the lungs including *pulmonary fibrosis*, *extensive tuberculosis*, *emphysema*, *pneumonia*, *pleural adhesions*, *neoplasms*, *hydrothorax*, *abdominal distention*, *ascites* and *hyperthyroidism*.

See *Differential Diagnosis of Bradypnea, Oligopnea and Apnea* (p 2014), *Hyperpnea*, *Tachypnea*, *Polypnea*, *Dyspnea*, *Orthopnea* and *Respiratory Irregularities* (p 2016).

DIAGNOSTIC ROENTGENOLOGY

Diagnostic roentgenology has been brought within the province of the office laboratory. The practitioner may purchase at a price approximating that of the inexpensive motor car a unit that can be set up in the office or transported to the home of the patient in a carrying case measuring 21 x 14 x 8 inches.

No special knowledge of electricity and physics is necessary. The radiograph is handled as easily as a modern automobile or a radio receiver. The danger of electrical shock is virtually absent. Complete protection is offered so far as radiation burns are concerned. Fluoroscopy may be carried out as simply as the projection of a moving picture film in the home.

PORTABLE X RAY SETS AND THEIR USE

The portable sets complete for radiography and fluoroscopy contain the following equipment:

- 1 Shock proof x ray tube head provided with a thermometer
- 2 Table tube stand for mounting the x ray unit
- 3 Compression cylinder which fits into tube head and permits accurate focusing and prevents scattering of the ray
- 4 Carrying case (21 x 14 x 8 inches)
- 5 Control units with voltmeter, milliammeter, kilovolt selector with three settings of approximately 5 kilovolts each, filament regulator which permits control of milliamperage through the range of the unit, an on and off switch, a bull's eye pilot light and an outlet for the connection of the foot switch or hand timer
- 6 A hand timer providing exposure interval settings of from $\frac{1}{8}$ to 10 seconds

this the film is washed again and hung to dry. It is quite possible with this arrangement to take the film, develop it and read it all within the span of ten or fifteen minutes.

The equipment requires little servicing. The expense of upkeep is negligible except for films and renewal of developer and fixer. The apparatus should last for a lifetime.

THE X RAY ROOM

Those practitioners whose demands cannot be met by the portable equipment can establish a radiographic room in the office laboratory. The space requirements are no more than those of an ordinary bedroom and it is a simple matter to black out the light and to arrange with the manufacturer of radiographic equipment for installation of wiring and machinery.

The techniques of radiography are no more difficult to master than those of the portable equipment. Each manufacturer furnishes directions and tables for simple and contrast diagnostic radiography and many provide teaching courses which can be taken within the duration of a week. Any physician who has learned to operate a motor car and a radio set can learn clinical radiography with slightly greater effort and application. The interpretation of findings comes only with experience and difficult films should be examined with the specialist roentgenologist. Abnormal fluoroscopic appearances also require consultation with the specialist.

are then made according to two tables provided by the manufacturer. One calls for a correction depending upon the reading of the voltmeter which will vary with the strength of the house current. The second reading provides the correct milliamperage and timing for the particular part that is to be filmed.

Two settings must then be made. The filament regulator is manipulated until the milliammeter is at the desired reading. The hand timer is then set for the required exposure. With the settings made, the tube head is placed

TABLE 197—CONTRAST ROENTGENOGRAPHY

| | Method | Reference |
|---------------------|--|--|
| Digestive System | Fasting barium meal | See Esophagus (p 1720) stomach (p 1743) and small intestine (p 1820) |
| | Cleansing enema barium enema | Large intestine (p 1830) |
| | Cholecystography (p 1989) | Gallbladder (p 2000) and biliary system (p 1989) |
| Urinary System | Excretion and retrograde urography pyelography and cystography | Kidney (p 2245) pelvis and ureters (p 2251) bladder (p 2252) |
| Respiratory System | Bronchography with iodide solution. | Bronchi (p 2061) and lungs |
| Nervous System | Air injections ventriculography and encephalography | Brain (p 1419) and cord (p 1430) |
| | Laminography | Spine (p 3003) |
| Circulatory System | Angiography | Peripheral vascular disease (p 996) congenital heart disease (p 956) |
| Reproductive System | Seminal vesiculography | Seminal vesicles (p 2254) |
| | Uterography | Uterus (p 2497) |
| Peritoneum etc | Pneumoperitoneum | Abdominal tumors (p 2621) |
| | Peri renal insufflation | Adrenal tumors (p 1978) |

at the required distance from the patient and an approximate focus is established. The part to be filmed is then placed on the cassette and the radiograph is taken by the simple device of pressing the switch of the hand timer.

Developing Radiographs—In the darkroom the film is removed from the cassette and fastened onto the film holder. It is then placed in the developing tank for five minutes at a temperature of 68° F. It is washed in water and placed in the fixer for a minimum of ten minutes. Following

this the film is washed again and hung to dry. It is quite possible with this arrangement to take the film, develop it and read it all within the span of ten or fifteen minutes.

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Associate Editor

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CLIFFORD R. SPINGARN, M.D.

CHAPTER 170

PRINCIPLES OF MEDICAL THERAPEUTICS

The armamentarium of medical therapy is rich in its variety and potentialities. The diverse modalities range from the herbals of the witch doctors to pure synthetic products from accurately measured pharmaceuticals to the no less potent instrumentalities of psychotherapy and correction of general hygiene. At times the physician functions as an arm chair advisor but he may participate in the more active procedures of manipulation, instrumentation, injection, aspiration or physical therapy. Most often he prescribes or administers drugs or biologicals for symptomatic, preventive or curative therapy.

Individualization in Therapeutics—In private practice the physician prescribes an individual therapeutic program for each of his patients. The regimen is modified by the nature of the ailment, the patient's way of life and capability for understanding, the cooperation of relatives and friends, the accessibility of apparatus and the practitioner's technical training and experience.

Individualization is the keynote of successful treatment. This axiom is dictated by principles of pharmacology, biology and psychology. The therapeutic task that confronts the practitioner is the treatment of the individual patient. Rules and experiences gleaned from the course pursued by any other patient serve only as general guides. Identical management for identical complaints will yield only a modicum of success.

The treatment of each symptom or disease is a new and fascinating experience with innumerable unforeseen variables. Complications arise, drug idiosyncrasy and tolerance are encountered, there are inexplicable changes for better or worse and unexpected recoveries and unforeseen fatalities arouse unreasonable hopes and justifiable fears.

The Execution of the Therapeutic Program—The therapeutic program cannot always be carried out by the physician without outside assistance. In outlying districts practitioners are compelled to be more self-reliant, whereas the urban doctor may employ a consultant and trained technician for each unusual procedure. Most country practitioners do obstetrics; few men in the cities are equipped to care for women in childbirth. Many rural physicians do minor surgery and perform the commoner major operative procedures.

In the home the physician may execute the orders or he may teach the simpler nursing procedures to the patient or members of the family. For the more complicated therapeutic techniques it is well to insist upon the services of a registered nurse, with transfer to the hospital as the alternative.

Therapeutic Equipment—The private practitioner is expected to provide himself with equipment for use in his office or the home of the patient. He must furnish himself with an emergency bag as well as apparatus

and other supplies necessary for the successful performance of the therapeutic program

TABLE 108—ESSENTIAL SUPPLIES AND EQUIPMENT FOR THE PRACTITIONER'S OFFICE

| | |
|---|--|
| Adhesive tape of 1, 2 and 3 inch widths conveniently threaded on rolls that fit into a wall bracket. Rolls of elastic adhesive plaster (in tin cans) and chiropodist plaster are stored in drawers or cupboards | Laryngeal mirror |
| Alcohol burner | Liquid adhesive |
| *Anesthesia mask | Loupe |
| *Applicators with cotton tips | *Lubricant both petrolatum and greaseless |
| Atomizers for oily sprays and aqueous solutions (vasoconstrictor and local anesthetic) | Magnifying glass |
| Bandage scissors | Obstetrical kit (p. 2077) |
| Basins | *Oiled silk for wet dressings |
| Battery jar fitted with perforated metal stand for the storage of syringes and needles. Keep on sterilizer stand | *Packing material in sterile tubes both plain and iodoform |
| Bottles glass stoppered with enameled labels for green soap, alcohol, ether, benzine, boric acid and iodine. Keep on sterilizer stand | *Poison antidote kit (p. 3318) |
| Carborundum stone for sharpening instruments and needles | Razor |
| *Catheters female metal or glass | Rubber bands for drains |
| Catheters male American synthetic with natural curve. Sterilize in 1% bichloride of mercury; do not boil | Rubber dams for drains |
| Catheters male plain rubber | *Rubber gloves |
| *Clinical thermometer | Splinting material light beaver board rolls and widths of plaster of paris and plaster splints |
| Cotton absorbent and non absorbent | *Stockinette for use under plaster |
| Dressing forceps in a glass jar containing phenol solution. Keep on sterilizer stand and use for handling sterile instruments | *Stomach tube with rubber aspirating bulb |
| Ear syringe | Rehfsuss bucket and Levine tube |
| Electric pad | Abbott tube (p. 1823) |
| *Finger cots | *Surgical instruments (p. 3309) |
| *Flashlight and batteries | Syringes and needles. The syringes should be all glass; they should include a 1 cc. tuberculin syringe with a blue piston, several 2 cc. syringes (one for aqueous solutions, one for oily solutions and one for metals such as bismuth, mercury and arsenic), a 5 cc., 10 cc. and a 20 cc. syringe. The needles should include the ordinary hypodermic needles both $\frac{1}{2}$ and 1 inch gauge, the deep injection needles of $1\frac{1}{2}$ inch, 18 gauge, intravenous needles $1\frac{1}{2}$ inch 20 gauge, a phlebotomy needle, a lumbar puncture needle. |
| Fountain syringe | Tape measure (metal) |
| Gauze sterile in roller bandages of various widths from $\frac{1}{2}$ to 4 inches. Separate sterilized packages for dressing (flat) 4 x 4 and 4 x 8 inches | Thoracentesis set (Lotan) |
| *Gauze adhesive bandages prepared for small dressings (Band Aids) | Stopper bottle and tubing |
| Head light | *Tongue blades |
| Head mirror | *Tourniquet |
| Hot water bottle | *Tracheotomy tube and air way (p. 3307) |
| Ice collar and cap | Tracheotomy set (p. 3357) |
| Intravenous sets. Store commercial sets of saline, dextrose in distilled water | Trochar and cannula for abdominal paracentesis (p. 1920) |
| Irrigation syringe all metal | Urethral sounds |
| Kidney basin | Urethral syringe all glass with rubber bulb |
| | *Vaginal speculum |
| | Vaginal tampons plain and medicated |

Carry also in emergency bag

Drugs and Biologicals—Drugs and biologicals are stocked for use in the office or in the patient's home. The supply may have to be duplicated, part being stored in a cabinet and the remainder in the emergency bag.

The extent of the store of *pharmaceuticals* varies in different offices.

TABLE 199—DRUGS AND BIOLOGICALS FOR THE PRACTITIONER'S OFFICE

| | |
|---|--|
| Acetic acid (2%) for white blood count test for albuminuria | Gentian violet solution (2 to 4% aqueous) |
| Acetylsalicylic acid (p 3837) tablets 0.3 gm (grains) | Green soap |
| Alcohol (9 %) | Holocaine hydrochloride (1% aqueous) |
| Aminophylline (p 3860) ampoules containing 0.24 gm (4 grains) for intravenous use | Homatropine (1%) |
| Apomorphine hydrochloride (p 3834) Hypodermic tablets 0.006 gm (1/8 gr) | Hydrochloric acid N/10 Hemoglobin estimation (p 3634) |
| Aromatic spirits of ammonia. Bottles and fragile ampoules | Hydrogen peroxide (3%) |
| Atropine sulfate (p 3875) hypodermic tablets, 0.001 gm (1/8 gr) | Hypnotic (p 3837) (Lustral secondamental) original capsules |
| Benedict qualitative solution for urine sugar (p 3674) | Insulin (p 1246) in vial U-40 contains 40 units per cc |
| Benzene | Iodine 2.5 and 7% Tincture |
| Benzoin compound tincture of to paint skin before use of adhesive | Liver extract for parenteral use (p 1077) |
| Bismuth preparation for intramuscular use (p 193) Council approved product | Council approved preparation |
| Caffeine sodium benzoate (p 3866) ampoules, 0.5 gm (7 1/2 gr) | Mapharsen (p 116) 0.06 gm (1 grain) for intravenous arsenical |
| Calcium gluconate ampoules (10%) for intramuscular or intravenous use | Mercury for intramuscular injection (p 2257) Council approved product |
| Carbolic acid small vial | Morphine sulfate (p 394) Mouth and/or hypodermic tablets, 0.008 and 0.015 gm (1/8 and 1/16 gr) and/or rubber stoppered vial containing sterile solution of which 1 gr = 1 cc |
| Chloral hydrate 0.3 gm (3 gr) tablets | Morphine substitutes (p 393) Dilaudid tablets 0.002 gm (1/8 gr) |
| Chloroform (p 3924) in dropper bottle | Nitroglycerin (p 3892) tablets 0.0006 gm (1/8 gr) Also amyl nitrite in pearls for inhalation |
| Codine phosphate (p 3854) Mouth and/or hypodermic tablets 0.015 0.03 and 0.06 gm (1/8 1/4 and 1 gr) and/or rubber stoppered vial containing a sterile solution of which 1 cc = 1 gr | Ointments Ammoniated mercury 5% Anesthine 10% boric acid 10% butacaine (p 3914) chrysarobin Leavitt's paste scarlet red sulfathiazole 5% sulfur ointment Lunatic acid 5% tar Whitfield's (p 3114) zinc oxide |
| Collodion flexible (59%) in ampoules | Plasma keep in ice-box |
| Demerol ampoules of 1 cc = 0.100 gm | Paraldehyde (p 3944) in bottle with rubber stopper |
| Dextrose ampoules 20 cc of 50% Also Sucrose or Sorbitol 50% in ampoules | Phenobarbital (p 3839) tablets 0.01 0.03 and 0.1 gm (gr 1/8 and 1 1/2) |
| Digitalis (p 833) oral and hypodermic preparations, council approved | Pentathol sodium (p 393) ampoules for intravenous use |
| Epinephrine chloride (p 3877) 1 oz. bottles of 1:1000 solution ampoules of 1 cc (1:1000 and 1:10,000) Bottle of 1:100 for spray | Phenobarbital sodium (p 3839) ampoules 0.12 gm (2 gr) |
| Ampoules of epinephrine in oil | Poison antidotes kit (p 3918) |
| Ethylmorphine hydrochloride (dionin) 0.5% for ophthalmic use | Posterior lobe pituitary preparations (p 1180) ampoules Also pitressin and pitocin |
| Epi edrine sulfate (p 3877) 1% capsules 0.60 gm (1 gr) | Procaine hydrochloride (p 3914) ampoules 1% and 2% |
| Ergonovine H Ampoules | Papaverine hydrochloride (p 3954) tablets 0.07 gm (1 gr) |
| Ergotamine tartrate (p 1309) Ampoules 0.0003 gm (1/8 gr) | Penicillin, ampoules of 100,000 Oxford units |
| Estrogen (p 2515) Council approved preparation in oil stilbestrol 0.002 gm (1/8 gr) | Physostigmine salicylate 1% |
| Ether (p 396) in can | Prostigmine (p 3874) ampoules 1:2000 1:4000 |
| Fluyl chloride (p 3926) in special glass vial | |
| Fluorescein 1% eye drops | |

Carry also in emergency bag

and in different localities. It is necessary to maintain a large number of preparations in smaller communities where there is not an adequate pharmacy; indeed in sparsely settled communities the doctor must dis-

TABLE 199 — DRUGS AND BIOLOGICALS FOR THE PRACTITIONER'S OFFICE (Continued)

| | |
|--|--|
| *Quinine hydrochloride (p 520) capsules 0.3 gm (> gr) | and powder sulfathiazole tablets and ointment (5%) sulfadiazine and sulfamerazine tablets 0.3 gm (5 gr) and 0.5 gm (7½ gr) |
| *Quinidine sulfate (p 861) capsules 30 gm (> gr) | Sodium sulfadiazine crystals in sterile ampoules |
| *Scopolamine hydrobromide (p 3875) tablets 0.0004 gm (1½ gr) | Streptomycin ampoules of 0.5 to 10 gm |
| *Silver nitrate sticks and solutions of organic silver (Silver) (p 134) Council approved | *Tannic acid solution 5% |
| Sodium chloride or Ringer's solution for intravenous use | *Testosterone propionate (p 2401) Council approved product in ampoules of 10 and 25 mg per cc |
| Sodium citrate (3.8%) for red blood count | *Vinyl ether (p 39 6) |
| Sulfonamides (p 88) Sulfanilamide tablets | Wright stain for differential count |

TABLE 199A — DRUGS AND BIOLOGICALS FOR THE PRACTITIONER'S OFFICE (Continued)

In Ice Box

| | |
|--|--|
| Crotalus Antitoxin (N.N.R.) for snake bite | Meningococcus Antitoxin (U.S.P.) for treatment |
| Diphtheria Antitoxin (U.S.I.) for treatment | Penicillin Units of 100,000 units |
| Diphtheria Toxin (U.S.P.) for Schick test | Rabies Vaccine (U.S.P.) for treatment |
| Diphtheria Toxoid Alum Precipitated (U.S.I.) for active immunization | Scarlet Fever Toxin for Dick test |
| Diphtheria Toxoid Tetanus Toxoid (combined) Alum Precipitated (N.N.R.) for active immunization | Scarlet Fever Antitoxin for blanching reaction and treatment |
| Diphtheria Toxoid Tetanus Toxoid Alum Precipitated Whooping Cough Vaccine (Combined) for active immunization | Smallpox Vaccine (U.S.P.) |
| Gamma Globulin for measles and infectious hepatitis prophylaxis | Streptomycin ampoules of 0.5 to 10 gm |
| Horse Serum for test of sensitization | Tetanus Antitoxin (U.S.P.) for treatment |
| | Tetanus Gas Gangrene Antitoxin (N.N.R.) |
| | Tetanus Toxoid Alum Precipitated (N.N.R.) for immunization |
| | Tuberculin Patches for skin testing |

* Should also be carried in emergency bag

TABLE 200 — CONTENTS OF THE EMERGENCY BAG

| | |
|---|--|
| Stethoscope | The electric diagnostic set (p 3583) or a battery |
| Sphygmomanometer | mirror head light nasal and ear speculums |
| Clinical thermometer | Vaginal speculum |
| Adhesive tape | Urethral catheters |
| Bandage scissors | A flashlight |
| An envelope containing oiled silk | Surgical instrument kit (p 3909) |
| 1 finger cots and rubber gloves | A container with syringes and needles |
| One strip of light beaver board for emergency use in the handling of a fracture | A compartment containing drugs listed in Table 199 |
| Stockinette | The obstetrical kit (p 2077) |
| One can plaster of paris (four inches width) | Tourniquet |
| Several packages containing sterilized gauze pads | Snake-bite set |
| Several roller bandages | Anesthesia mask |
| Tongue blades | Glass slides for spreads |
| Wooden applicators with cotton tips | Blood counting set (p 3674) |
| A lubricant | Test tubes |
| Several tubes containing sterile packing material (plain and iodoform) | Tracheotomy tube |
| | Stomach tube |
| | Poison antidote kit (p 3318) |

pense as well as prescribe. Men who practice in large cities where drug stores are more numerous require a minimum number of preparations for emergency use.

In the case of biologicals it is difficult to decide between safety and

economy since to stock a complete supply is to incur considerable expense. On the other hand delay in securing the needed biological may mean the loss of precious time and jeopardy to a human life.

The preparations listed in Table 199A should be checked by each physician to determine his own particular need. They represent an irreducible minimum for routine use by the rural practitioner and for emergency use by the city practitioner.

The Emergency Bag—The practitioner must be prepared to deal with exigencies in the patient's home. The emergency bag contains equipment and supplies for this purpose. It should have a variety of compartments so that its contents do not become hopelessly messed about in the course of travel. Two bags should be kept in readiness, one to remain in the office in a state of completeness and neatness and the other to be left behind at the end of the day for renewal and preparation.

Many of the materials used in the emergency bag may be put in at the last moment so as to save the expense of buying supplies in duplicate. Thus the electrical diagnostic set, the blood pressure apparatus, the tuning fork, the reflex hammer, and the kit of surgical instruments are picked up when needed and returned to the office stock when the practitioner gets back from his daily rounds.

CHAPTER 171

THE PHYSICIAN AS AN ADVISOR

INTEGRATIONS

Psychotherapy (p 1327)

Dietotherapy (p 665)

Nutrition and Diet (p 585)

The American Dietary (p 633)

Nutrition and Metabolism (p 691)

Diets in Health and Disease (p 638)

THE physician functions as an advisor and armchair philosopher during some part of each consultation. In this guise he deals with psychotherapy or general hygiene; he explains the natural course of the disturbance or disease under consideration; he prescribes rest or measures for restoration of function; he suggests occupational therapy, a vacation, climatotherapy, balneotherapy, spa therapy, or a change in diet; he teaches a nursing technique to the patient or a relative; or he leaves orders for the professional registered nurse.

PSYCHOTHERAPY

Psychotherapy is the art of treating mental disease or disorders; any measure, mental or physical, that favorably influences the mind or psyche. Wittingly or not, each physician is a psychotherapist. Each patient who receives any type of treatment for whatever cause receives an additional measure of psychotherapy. For the great masses of the people, the family doctor is the only psychotherapist to whom there is access; in this capacity, he dispenses encouragement, understanding, and sympathy, and explores and attempts to simplify the mundane problems that give rise to the conflicts of daily life.

Because of its important significance, nontechnical psychotherapy (p 1316) has had previous extensive considerations which the reader is urged to give his most careful study. The more formidable methods of psychotherapy which are required in the treatment of the *psychoses* (p 1364) and the obstinate and incapacitating *neuroses* (p 1335) are the domain of the psychiatrist.

DIETOTHERAPY

The important material on dietotherapy is considered in the section on *Metabolism* (p 585). The presentation of practical diets in health and disease is preceded by a discussion of the foodstuffs that make up the American dietary and by a review of the extraordinary metabolic processes of which the human body is capable.

The extent of undernutrition, malnutrition, and deficiency states throughout the world makes it imperative that the practitioner be thoroughly conversant with food requirements, food preparation, the symptoms of such deficiencies as the *avitaminoses* (p 616) and the means by which these conditions can be prevented and alleviated.

CORRECTION OF GENERAL HYGIENE

The correction of general hygiene without alteration of the environment of the patient is a most important therapeutic procedure. Many symptomatic complaints without organic basis result from an ignorance of the rules of normal living. The effective therapeutic measure is not the administration of symptomatic medication but the removal or correction of the etiologic factor or factors.

Advice concerning general hygiene may be prophylactic or therapeutic. For example, many wise parents make it a point to bring their children to the physician for a discussion of *sex hygiene* (p. 2401). *Pre-marital conversations* (p. 2400) are highly efficacious for prospective brides and grooms. It is amazing to note the biological ignorance of even the most sophisticated of the younger generation. A great deal of difficulty can be avoided by discussions of the *physiology of pregnancy and lactation* (p. 2617) with the expectant mother. Young matrons may be instructed concerning the *prophylaxis of the exanthematous diseases in childhood* (p. 80). Housewives often need advice concerning *diet* (p. 633). Boys and young men should be taught *sexual hygiene* and if necessary *venereal prophylaxis* (p. 3122).

Correction of general hygiene may be curative. Many headaches result from constipation, over-indulgence in alcoholic drinks, eyestrain or dietary errors. Probably the majority of gastric complaints follow abuse of a perfectly normal stomach. Insufficient sleep, fatigue, anxiety and worry give rise to widespread disorders in the realm of the involuntary nervous system. Bad shoes, improper corseting and faulty body mechanics produce more symptoms referable to the skeletal system than rheumatic fever.

It is the function of the practitioner to teach habits of health. He should attempt to banish the association of the physician with illness. The patient should not consult a practitioner with fear and reluctance—so well illustrated by the mother who drags a screaming child to the doctor's office. A better attitude is that of the patient who arrives with a certain amount of eagerness and interest.

Instruction in general hygiene must not be made too academic. Complicated terminology is avoided. The vernacular is better employed. Preaching is to be shunned and hypocrisy is intolerable. Not much can be accomplished by the physician who preaches good hygiene and practices bad hygiene. It is of course relatively useless for the physician who is addicted to alcohol or tobacco to preach to the patient that these habits are noxious or harmful.

VIS MEDICATRIX NATURAE

The great majority of human ailments tend towards spontaneous recovery. The intelligent doctor humbly recognizes the healing art of nature. He learns to avoid interference with the favorable course of disease. But he does not neglect the patient, belittle the illness or the symptoms or fail in his attempt to assuage subjective complaints.

Skilful Neglect.—In self-limited diseases if specific therapy is not indicated the regimen may be one of skilful neglect. Intelligent patients are told that recovery is certain and that treatment is unnecessary. The

more ignorant should be given symptomatic therapy since they cannot be expected to understand that reassurance alone will suffice

REST

Rest is one of the most useful therapeutic measures. It ranges from peace and sanctuary to a plaster cast and is a difficult prescription to dispense. It travels hand in hand with the spontaneous course of healing, and is its greatest adjuvant.

Rest at Home—Hospitalization solely for the purpose of securing rest, is usually impracticable. In most instances compromises are sought. A wage earner with the cooperation of the family may be able to go to bed immediately upon his return from his labors and spend the ensuing twelve to fifteen hours at fairly complete rest. The housewife may obtain partial rest by having a few hours of relaxation in the morning or afternoon when the family is away.

It is sometimes possible for the patient to spend the weekend in bed or to have an additional "cure" day in the middle of the week. The "cure" day should be regimented by prescribing a saline laxative in the morning, sedatives during the day, a hypnotic at bedtime, and a warm bath or shower in the afternoon. Wealthy patients may be indulged further by massage.

Rest Cure in the Hospital—If patients are able to gild their infirmities, the *Weir Mitchell rest cure* may be of great benefit in the exhaustion and fatigue states.

The patient is isolated and confined to bed. The underweight are given forced feedings, the obese are fed low caloric diets, tobacco and alcohol are banned, the colon is well cleaned, the relaxation tub, pine needle bath or the Scotch douche help to fill out the day. Massage and exercise provide tonic care. Reading and occupational therapy are encouraged and the radio is a useful adjuvant, provided it is used on schedule and not permitted to drone on through the day.

The rest cure should be continued for at least ten days and preferably three weeks. The patient is warned that the fatigue symptoms are often intensified and accompanied by depression on the third or fourth hospital day. Anticipation of this eventuality prevents the inpatient from checking out prematurely.

Rest Cure by Transplantation—A geographic change is a more virile method of obtaining rest and an escape from the troubles and cares of the world. A fishing or a hunting trip, sailing or golfing may accomplish the therapeutic purpose.

The *escape factor* is encouraged by the various spas (p. 3764). Credit for the improvement is usually attributed to the climate, the altitude, the air, or the drinking water. In all likelihood the factor of escape is the more important, the physical factors being of distinctly minor benefit. Oftentimes the further the place of cure from home, the greater its virtue, the greater the element of mysticism, the more potent the cure. Hence spas in other countries excel native institutions.

The knowing physician may reasonably become party to a psychological plot to assist the patient in his escape by prescribing a cure which

places distance or in normal times even the ocean between the sufferer and his situational difficulties. So long as this is done with tongue in cheek and the favorable result not attributed to heliotherapy or radio active waters and so long as no economic strain is placed on the shoulders of the patient or particularly his supporters no great harm is done and much benefit may ensue.

Rest of Organs by Immobilization—Rest of a part of the body is of frequent benefit. Examples include the plaster cast for a fractured bone or an induced pneumothorax to collapse a tuberculous lung.

The Dangers of Rest—The prescription of rest in bed has however disadvantages and dangers. The patient may exploit the remedy at the expense of family, community or his own initiative. Those with anxiety states develop the fixed idea that they must be afflicted with some ominous disease of whose presence they are being kept in ignorance. During prolonged immobilization as in the case of a fractured limb the part becomes less vascular, blood flow is decreased, tonicity is diminished, overlying skin and underlying muscle undergo atrophy and the required immobilization of bone is attained at the expense of the efficiency and integrity of the soft parts (p. 4117).

When the whole body is placed at complete bed rest as in cardiac invalidism or a prolonged surgical incident more profound and important dangers are encountered. In addition to restlessness and mental depression the patient notes insomnia, anorexia, constipation, impaction of feces and difficulty in urination. Bed sores are invited and the hazards of intravascular thrombosis and subsequent embolism hang over the victim like the sword of Damocles. To prevent the evils of the rest cure the patient is encouraged to move legs and arms at regular intervals, a bedside commode is introduced, lavatory privileges are given as soon as possible and deep breathing exercises are conducted several times daily (p. 4127).

RESTORATION OF FUNCTION

Following immobilization or the rest cure the physician encourages his patient to prepare for the resumption of normal activities. There is a growing and commendable tendency toward shortening the period of invalidism following acute infections, trauma and operative procedures. Many surgeons order the patient out of bed three to five days after appendectomy compared to the ten to fourteen day period that formerly prevailed. This practice has unquestionable merit in preventing the dangers of prolonged immobilization (p. 4121).

The decision to terminate bed rest in insidious and recurrent infections such as *tuberculosis* (p. 268) and *rheumatic fever* (p. 186) presents greater difficulties. Premature resumption of activity may result in a protracted recrudescence for which the enthusiastic physician is held culpable. Amongst other factors which the practitioner must consider in arriving at his conclusion is the temperament of the individual patient. A high powered executive and a conscientious housewife may seize the initiative if their medical advisor exerts hyperprecaution; on the other hand the mild mannered and the mollusc may have to be driven from an under cover existence and literally forced into activity.

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The knowing physician may reasonably become party to a psychological plot to assist the patient in his escape by prescribing a cure which

Simple calisthenics and dancing are recommended highly for the otherwise healthy sedentary worker. The American Medical Association publishes an inexpensive pamphlet describing exercises for the business man and the business woman. These do not require apparatus. They are simple and well illustrated. Many patients try home exercises with dumb bells, wands, punching bags and rowing machines, but these are usually transitory fads. Regular attendance at the gymnasium involves excessive expenditure of time and energy; the midday exercise period is open to serious criticism since the patient must undress, exercise, bathe, dress and then return to his labors. The tension involved more than cancels the possible benefits.

Many city dwellers indulge in an orgy of athletics over weekends or holidays. This spasmodic athletic career is usually expensive and results in local trauma, blisters, pulled tendons, a charley horse, sprains and contusions. Contact games such as football, basketball and even baseball are probably better abandoned after the thirtieth year unless they are followed as a profession. Tennis may be enjoyed for a considerably longer period though it would seem unwise for the enthusiasts to play singles beyond the thirty-fifth year. Tennis doubles seems to have no particular age limit and may be continued as long as the player experiences no unusual strain or fatigue.

The ideal sports for the older man or woman are sailing, gardening and golf. The last is effective therapeutically for the player whose prime object is a pleasant excuse for taking a stroll over rolling countryside. The golfer who seeks for perfection often forgets the intended purpose of his game and plays competitively and under strain against his rival or against par; such misguided recreation may be detrimental to health.

Graded exercises are of great value for the cardiac invalids. The spa at Saratoga in New York provides graded walks which vary in length and degree of inclination; there is also a nine-hole golf course with a maximum grade of 5 degrees.

CORRECTIVE EXERCISE

Corrective exercise is of value particularly for patients with poor bodily mechanics, scoliosis, flaccid and spastic paralyses, peripheral vascular disease or *tuberculosis dorsalis*. The standard exercises for correction of posture deformities of the spine, flat feet and low back difficulties are described in the Bulletin of the Children's Bureau of the United States Department of Labor (Publication No. 1650).

These exercises should be started in recumbency. One of their purposes is to enlarge the chest capacity by increasing the excursion of the diaphragm and strengthening the other muscles of respiration. With every contraction of the diaphragm, the liver, the stomach, the small intestine and the transverse colon move upward and downward. A type of internal abdominal massage is thus induced.

The exercises also improve the tone and increase the power of the anterior and lateral abdominal muscles, the glutei and the spinal group. The healthy condition of these muscle groups is essential to the acquisition of proper posture.

CONVALESCENCE

The period of convalescence is the gap between necessary immobilization and restoration of normal function. Individuals vary in the resiliency of their tissues. The young and robust possess the elasticity of a golf ball; frail and delicate patients, as well as the older people, make only a gradual comeback (p. 4117).

In general, the period of convalescence equals the period of active disease. Premature resumption of a full schedule often results in obstinate malaise and inefficiency.

PASSIVE MOTION

Passive motion places the least strain on the patient who is attempting to resume normal functions. To accomplish this step on the road to recovery, the physician puts each joint through its range of normal activity.

RESISTIVE MOTION AND PASSIVE EXERCISE

Resistive motion requires the cooperation of the physician. The patient performs voluntary movement while the practitioner acts the role of the antagonist. Only sufficient resistance is employed to hinder but not completely to frustrate the voluntary movement. Resistive exercise is of great value to the cardiac patient who must be confined to bed for a prolonged period.

Schott Exercises—The Schott exercises recommended for the rehabilitation of the cardiac invalid are essentially those of passive motion. The legs and arms are placed through the ranges of their normal arcs of motion. Slight resistance is offered by the medical or nursing attendant and each movement is so timed that its completion takes no less than one-half minute. A rest period of one-half minute is interpolated before the next movement is begun.

The practitioner lends the security of his presence to the first exercise or activity of the convalescent. He notes the response to physical exertion, particularly the effects on the pulse rate and the rate and depth of respiration. No form of activity is permitted that is productive of significant dyspnea or tachycardia.

PHYSICAL EXERCISE, GAMES AND RECREATIONS

Active restoration of function is accomplished by corrective or recreational exercise, occupational therapy, games and recreation. The Greek ideal of the sound mind in the healthy body is one to which all physicians subscribe.

In city practice, particularly, the practitioner is harassed by the miscellaneous ailments of the sedentary. Sooner or later the typical office worker develops low-grade symptoms: the arches flatten, the back becomes weak and painful, constipation is the rule rather than the exception, headaches are frequent, there is somnolence in the afternoon and insomnia at night, weight increases, resistance becomes diminished, the upper respiratory infections occur frequently and persist for prolonged periods, the reaction to disease and surgical procedures is sluggish, and minor medical and operative incidents become major and formidable problems.

Simple calisthenics and dancing are recommended highly for the otherwise healthy sedentary worker. The American Medical Association publishes an inexpensive pamphlet describing exercises for the business man and the business woman. These do not require apparatus. They are simple and well illustrated. Many patients try home exercises with dumbbells, wands, punching bags and rowing machines, but these are usually transitory fads. Regular attendance at the gymnasium involves excessive expenditure of time and energy; the midday exercise period is open to serious criticism since the patient must undress, exercise, bathe, dress and then return to his labors. The tension involved more than cancels the possible benefits.

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Exercise II—Starting Position—Sitting—hands on head

Exercise—Raise one shoulder elevating ribs—hold this upward stretch position and then contract side abdominal muscles Alternate

Exercise III—Starting Position—Lying on right side—stretch arms forward, shoulder high—flatten the lumbar curve by contracting the abdominal and gluteal muscles Raise right leg upward—lower—repeat the same on other side

Exercise IV—Starting Position—Lie over edge of table face down—feet on floor—stretch arms over head and grasp sides of table—raise legs backward on level of spine—heels together

Exercise—Carry legs to right—then left—continue alternating sides

Exercise V—Starting Position—Standing—hands on head—flatten the lumbar curve and stretch up ribs

Exercise—Bend trunk left—repeat same to right

ROUND SHOULDERS

Exercise I—Starting Position—Lie over edge of table—face down—feet on floor—clasp hands behind back

Exercise—Roll shoulders backward

Exercise II—Starting Position—Standing—clasp hands behind back—flatten lumbar curve—chest well over toes

Exercise—Roll shoulders backward

Exercise III—Starting Position—Stand facing corner of room Place hands at shoulder height against walls—fingers pointing inward

Exercise—Rise on toes—press hands against wall and allowing elbows to bend force chest into corner—keeping elbows and shoulders high

Explanation—During this exercise it is most important to keep the low back flat Do not allow the hips to sag forward This conscious motion should be at the shoulder girdle A strong pull is felt across the chest

FOOT EXERCISES

Exercise I—Starting Position—Sitting on table—feet raised slightly from floor—toes pointing inward

Exercise—Curl toes

Explanation—Curl toes under as if grasping a small marble

Exercise II—Starting Position—Sitting on table—raise feet slightly from floor—knees straight

Exercise—Foot circumduction

Explanation—Point toes downward inward upward (never outward) When drawing feet upward curl the toes under as much as possible to raise the arch

Exercise III—Starting Position—Standing—hands on hips—feet parallel one half inch apart

Exercise—Roll weight on outer border at the same time curling toes under

Exercise IV—Starting Position—Sitting

Exercise—Curl toes under and pick up small marble from floor

Exercise V—Starting Position—Sitting—place a tape under the ball of the foot, holding end of tape in each hand

Exercise—Pull strongly on tape so as to flex the ankle to stretch the Achilles tendon while voluntary effort is made to flex the foot dorsally

Explanation—While stretching the tendon the toes should point inward

Exercises for use in *flaccid and spastic paralyses* are too technical for the average practitioner The patient who suffers from either of these conditions should be referred to an expert

OCCUPATIONAL THERAPY

Occupational therapy is ordinarily limited to those institutions as yet far too few that are properly equipped and staffed for this important work

Needle and textile work, basket weaving, leather and metal craft, book binding, carpentry, horticulture, dancing, printing and the simpler applications of the fine arts are taught by experts and practiced by the convalescent, disabled or handicapped patient. The efficacy of occupational therapy and its value in rehabilitation, both mental and physical, cannot be overemphasized by even its most enthusiastic advocates (p. 4119).

In private practice the physician must not neglect occupational therapy though he may be unqualified to teach any of the special technics. Even more important than the technical procedure is the beneficent and happy effect upon morale. Women patients while in bed or semi-invalided may be encouraged to do needle work. Those who have any musical training should resume their practice at the earliest possible date. Men should be urged to perform manual tasks even though the pattering serves no useful purpose. The ambitious intellectualized individual is particularly benefited by manual therapy which has no practical gain.

If careful inquiry is made it is usually found that the bedridden or incapacitated patient has some artistic or occupational resource. Often times even in the most unlikely candidates there will be discovered a genuine talent for some unexpected technic such as sketching or painting.

THE VACATION

The vacation should be regarded as a period of convalescence. Its purpose which is usually lost among other considerations is to secure rest and a change as an escape from routine.

In most instances the vacation is so badly planned that the convalescent returns to his labors more fatigued than on departure. It is for this reason that the practitioner should attempt to assist his patient in the selection of the vacation. Parents rarely enjoy much relaxation when they holiday with their small children. Many men enjoy a marital vacation in the companionship of fellow fishermen or hunters. It is sometimes better for the head of the family to remain at home in quiet and solitude while his dependents are scattered in the pursuit of happiness.

CLIMATOTHERAPY

Climate and weather produce a profound effect upon the human organism and upon pathological conditions. Each of us is aware of differences in well being dependent upon *altered weather conditions* of temperature and barometric pressure. On a broader scale there are *seasonal effects* upon physiologic processes, the incidence and severity of disease. There are obvious differences between the inhabitants of the torrid, temperate and arctic zones.

For the general practitioner the implications of climate have mostly academic biologic significance. His problem of course is the care of the individual patient under the climatic conditions that exist in his peculiar and particular locality. In a general way he observes that epidemics of the common upper respiratory infection occur in the Fall and in the Spring. Gastro-intestinal disorders and the fevers of enteric origin are more prevalent in the Summer.

A great deal of nonsense has been written concerning climate, particu-

larly in continental Europe. The cure doctors recommend specific localities for the relief of anemia, gallstones, renal concretions, bronchitis, the rheumatic difficulties or cardiovascular phenomena. The alert practitioner realizes that these pronouncements are merely designed to lure the naïve whose bank account is disproportionately large in relation to the fund of medical information.

Demonstrable Climatic Effects—In more recent years students of the effects of climate have demonstrated that *rheumatic fever* is more common in the cities of the Northern and Midwestern United States than in the Southern states. A rheumatic child, leaving the temperate zone and moving to a climate such as prevails in Florida, New Mexico or Arizona may be quite free from infectious manifestations only to experience these again upon return to unfavorable climatic conditions (p. 193).

Similar but less tangible claims have been made for the climatic control of respiratory infections and the infectious types of *arthritis* (pp. 290, 291).

Variables in Evaluation of Climatotherapy—At times the beneficial effect attributed to climate is due to some other factor. While it might appear that goiter occurs in certain regions, the actual etiology of the thyroid enlargement is the deficient iodine content of the soil and water. Correcting the iodine deficiency reduces the incidence of goiter while climatic conditions are unchanged. The allegedly beneficial effects of climate on diabetes mellitus are probably due to the social and psychological alterations that have occurred in the transplanted patient.

Indications for Climatotherapy in Private Practice—In private practice the advisability of climatic cure arises in the following conditions: (1) *rheumatic fever*, (2) chronic or recurrent *upper respiratory infection* (sinusitis), (3) chronic respiratory infection such as *bronchitis*, *emphysema* and *bronchiectasis*, (4) *tuberculosis*, (5) chronic or recurrent infections or atrophic *arthritis*, and (6) *debilitated patients* who suffer from chronic disease, particularly cardiovascular degeneration.

Practical Aspects of Climatotherapy—In arriving at his decision concerning climatotherapy the physician must take into consideration a number of practical circumstances:

1. The patient must be prepared for an *environmental change*.

2. There is considerable *expense* involved in closing the home, traveling to the approved climate and re-establishing living quarters. A wage earner must give up occupation and seek another job. An employer must be prepared to leave his business or sell or abandon it and establish himself again in the new locality. Dependents impose a financial burden on the stay at homes.

3. If and when recovery takes place and the patient has become acclimatized, the risk of the return to the original locale is fraught with the dangers of *recrudescence* and *remission*. It is a common experience in temperate climates to observe respiratory infection arise within a fortnight after the northward migration of the patient who has just returned from a southern vacation.

4. If the patient leaves the family circle and attempts alone to seek a climatic cure, there are other unfavorable features. The sufferer must depart from his bed and board, the care of the family and the practitioner and dwell in an institution or a residence hall. The atmosphere is strange

The food supply is different The companionship and advantages of the home are *dislocated*

It may not be long before the hope of the voyage is replaced by an aching nostalgia

A few practical generalities concerning climatotherapy are therefore warranted (1) Climatic cure should not be attempted in terms of days or weeks It must be employed over a span of *months or years* (2) climatotherapy should not be attempted until and unless the *course of the diseased process is unfavorable* despite best efforts in therapy under prevailing conditions (3) climatotherapy must not be suggested if the *economic sacrifice is so great* as to upset the equilibrium of the patient and his family (4) climatotherapy must be of urgent moment if its adoption means *separation of marital partners children and parents* or the breaking up of a home (5) *there is no type of climate that cannot be obtained in the United States*

Climatotherapy in the Home — Certain climatic boons may now be brought to the patient thanks to the great engineering advances of recent years *Heliotherapy* can be best administered in measured dosage with complete accuracy by means of the various forms of *ultraviolet irradiations* The machine purchased should be a type approved by the Council on Physical Medicine of the American Medical Association (p 3794)

Regulations of temperature and moisture and exclusion of dust borne irritants are best obtained by means of an *air conditioning apparatus*

The patient who dwells in his own home at an expense that is relatively minor compared with the cost of climatotherapy may secure the benefits of heliotherapy optimum temperature moisture and dust conditions without ever leaving the confines of his bed chamber

BALNEOTHERAPY

Balneotherapy comprises treatment by the internal and external use of mineral waters The principal constituents of these waters are sodium magnesium calcium and iron in the form of chlorides sulfates sulfides and carbonates Some waters are reputed to contain minute quantities of radium Curative waters may be hot warm cold carbonated or flat The advocates of balneotherapy commonly stress the fact that the waters are *natural* hence possessed of mysterious biological qualities To obtain the natural water the patient must go to the source of the supply In most instances this source is a spa that has the unbiased recommendation of the local board of trade and the spa physician

The waters may be taken *internally* as any other antacid or saline laxative or they may be applied *externally* particularly if they are hot and contain sulfur for their reputed effects on the skeletal system Except for the psychological factors of environmental change and escape it is doubtful whether anything is to be gained by the external or internal use of the balneotherapeutic preparations other than what might be obtained from their similar use employing bottled preparations for internal administration and the home bathtub for external preparations (p 3132)

Mineral Springs in the United States — The popular mineral springs in the United States are at Arrow Head California Hot Springs Arkansas Hot Springs Virginia White Sulphur Springs West Virginia Warm Springs

Georgia French Lick Indiana Sharon Springs Glen Springs and Saratoga in New York Mount Clemens Michigan, Martinsville Indiana Poland Springs Maine Mountain Valley Arkansas, Waukesha Wisconsin

Balneotherapeutic Modalities—At the mineral springs the following clinical applications of the balneotherapeutic agencies are available

1 *Subthermal baths* are given at temperatures varying from 90 to 98 Fahrenheit These are used to soothe irritability of the nervous system

2 *Thermal or resolvent baths* are given at temperatures of from 90 to 104° Fahrenheit They are suggested for the treatment of painful conditions of the nerves muscles and joints They relieve pain decrease spasm and increase circulation of an affected part

3 *Hyperthermal baths* at temperatures of 104 to 130° Fahrenheit can be given for only a few moments They are usually applied locally in the form of sitz baths for the treatment of chronic inflammatory pelvic disease

4 *Vapor baths* utilize the steam that arises from the thermal mineral springs in a vapor room or cabinet They are recommended for the treatment of painful conditions of the skeletal system

5 *Sulfur baths* may be taken by the hardy or zealous anosmic for the treatment of chronic skin conditions and rheumatism

6 *Effervescent baths* containing carbon dioxide are of the type popularized at Bad Nauheim They are used in the treatment of cardiovascular disorders It is difficult to understand how any significant therapeutic purpose can be accomplished by this form of therapy At most a transitory change in pulse rate or blood pressure is demonstrable For the rest the benefits are probably mere wishful thinking

7 *Peat or mud baths* are employed for skeletal disorders Children have been punished for less misconduct

8 Inhalation of various mineral waters is recommended in the treatment of chronic respiratory functions Chemically the inhalants contain alkaline chloride mixtures carbon dioxide or hydrogen sulfide

9 *Sea bathing* (thalassotherapy) has been recommended for the relief of arthritis and peripheral vascular diseases Certainly it can do no harm and is pleasurable if nothing else

SPA THERAPY

The spa is the health resort at which the patient indulges in dietotherapy balneotherapy climatotherapy and hydrotherapy The specific beneficences of spa therapy include (1) changes in climate or atmosphere (2) the presence of natural earth and waters for internal or external use (3) the employment of natural steam and vapors for inhalation and (4) the simultaneous prescription of hydrotherapy physiotherapy and dietotherapy

A most important therapeutic adjuvant of spa therapy is the fact that the patient has been permitted to indulge in the *escape mechanism* by leaving his normal environment An accessory valuable factor is that here the patient follows a therapeutic regimen that would probably not be obeyed at home

There is a certain type of patient notably seen among the wealthy coddled class who will enjoy a splendid therapeutic result on removal to the spa Such patients include the fatigued man of affairs with his multi-

phicity of responsibilities and cares. It is quite impossible for him to care for his general health while in the midst of his routine turmoil. Similarly, the indulged woman may temporarily follow the normal rules of general hygiene. She will get a certain amount of physical exercise, indulge in massage, follow a diet, get to bed at a reasonable hour at night and abstain from alcohol or cigarettes. If she has dieted herself excessively, she regains needed weight. The gluttonous may practice auto cannibalism by living on their own blubber.

The practitioner employs the spa for his recalcitrant patient who can not or will not follow a general health program under any other circumstances. If there is more value to spa therapy than is indicated above, it is certainly not demonstrable to the vast body of practicing physicians. The villagers who dwell in the vicinity of the spa rarely if ever indulge in the facilities that attract the tourist. Perhaps it is that when they seek spa therapy, they go to a distant health resort!

CHAPTER 172

TREATMENT BY MANIPULATION

MANIPULATIVE treatment is as ancient as the practice of medicine. Besides its physical components it has psychogenic implications which require consideration in estimations of therapeutic efficacy. The mere laying on of hands has ritual significance in all theologies. The child clings to the parent's hand for security and guidance. fraternal organizations have peculiar handclasps. lovers proverbially hold hands and the physician often pats his patient's hand by way of assurance. Physical contact through the mediation of the hand appears to convey the thought that the spirit has reached to the flesh.

MASSAGE

The varieties of massage include *effleurage* or stroking, *friction*, *petrissage* or kneading and *tapotement* carried out by clapping, striking or vibrating the tissues.

General Massage—General massage is prescribed in the treatment of convalescents. It is also a luxury widely employed by coddled individuals of sedentary habits whose physical activities are limited to the muscle contractions required for the ingestion of food and the performance of essential biological functions. Many obese patients employ massage for the purpose of rubbing off fat. the resultant changes in dimensions are more likely due to uneven tension on the tape measure. Nervous, highly strung patients frequently are relieved of insomnia by *effleurage* taken just before retiring.

Local Massage—Local massage has demonstrable value in hypoplasia and atrophy of an extremity such as results from disuse due to immobilization or poliomyelitis. *Effleurage* improves the local circulation, *friction* relieves the indurative types of myositis and aids in the dissipation of infiltrations and thickenings in bursae, tendons and the fascia.

Evaluation of Massage—There is no objective method of evaluating the benefits of massage. Patients frequently report a sense of well being. Often this is greatly influenced by the lingual gymnastics of the rubber whose loquacity often discourages the physician from greater employment of his services.

The strongest argument that favors massage is the attitude of athletes in training. Most competitors utilize a masseur before and after participation in games or sports. Professional baseball and football players submit to a rubdown to limber up before a contest and to restore their muscles at the end of the contest.

ARTIFICIAL RESPIRATION AND RESUSCITATION

Measures for artificial respiration and resuscitation are required in the treatment of acute or chronic respiratory failure.

Prone Pressure Method—When instrumental devices are not available the Schaefer prone pressure method is employed. The patient is placed on the ground face downward with forehead resting on forearm to insure patency of nose and mouth. The physician straddles the recumbent patient and brings on forced expiration by compression of the lower ribs. With release of pressure the recoil from the position of forced expiration induces inspiration.

Rocking Seesaw Method—The rocking seesaw technic possesses certain advantages over the Schaefer method. The patient is put on a board or stretcher which is pivoted at the middle on a fulcrum. By rhythmic rocking in seesaw fashion the weight of the abdominal viscera pushes the flaccid diaphragm up and down.

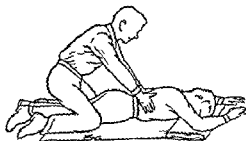


Fig. 1102.—Release of pressure on inspiration in the prone pressure method of artificial respiration.



Fig. 1103.—Application of pressure on expiration in the prone pressure method of respiration.

Pulmotor—The more serious respiratory depressions require the use of the pulmotor which may be obtained in most operating rooms from the local fire or police departments or from the public utility companies. The practitioner should anticipate the emergency by ascertaining the exact location of the pulmotor before any acute exigency arises.

The Iron Lung (Respirator)—The iron lung is a mechanical respirator that may be used as a temporary expedient or for protracted respiratory paralysis such as occurs following the bulbar type of *poliomyelitis* (p. 460).

The Drinker model is a cabinet that encases all of the patient's body except his head. The respirator is rendered airtight and the chest is expanded by reduction of the pressure within the apparatus; the natural elasticity of the lung is responsible for the expiratory process. Patients

may be kept alive in a respirator for months or years but a trained attendant should be kept in constant attendance

MANIPULATIVE SURGERY

Until recent years manipulative surgery occupied a province outside of general medicine where it was conducted primarily by bone setters. Osteopathy and chiropractic have been natural developments whose popularity attests to the therapeutic efficacy of non operative surgery.

Manipulative surgery is of value in the reduction of dislocations and subluxations (p 2964). It is also useful in dissipating peri articular adhesions (p 2894) and indurations in muscle and fascia. Many of its most dramatic triumphs are in functional or hysterical contractures (p 2948).

CHAPTER 173

TREATMENT BY INSTRUMENTATION

THE natural body orifices which include the nose mouth ear urethra vagina and anus lend themselves to instrumentation Through them the practitioner performs treatments by lavage aspiration irrigation and dilatation In certain instances the ease with which the technical procedure can be executed places it in the category of a nursing procedure as illustrated by irrigations of the nose throat and ear The passage of the stomach or duodenal tubes urethral catheterization in the male and irrigation of the bladder are performed by the practitioner specialist consultation is required for the more difficult operation of dilatation of the esophagus

The various instrumental procedures are most conveniently described with the clinical abnormalities of the organs concerned Thus washing of the stomach and duodenum irrigation of the colon and enemas are described in the section on the digestive system vaginal douching and the use of pessaries are taken up with the consideration of the female reproductive organs References to source material are as follows

| | |
|---|-----------------------------------|
| Dilatation of Esophagus (p 1725) | Nasal Instillation (p 2027) |
| Gastric Lavage Gavage and Drainage (pp 1749 1751) | Nasal Spray (p 207) |
| Milk Drip (p 175) | Steam Inhalation (p 2028) |
| Duodenal Drainage and Lavage (p 1752) | Ear Irrigation (p 208) |
| Intubation of Small Intestine (p 1823) | Catheterization of Female (p 235) |
| Dilatation of Rectum (p 1810) | Catheterization of Male (p 236) |
| Enema (p 1824) | Urethral Irrigation (p 2335) |
| Colon Irrigation (p 184) | Urethral Dilatation (p 234) |
| Proctositis (p 1825) | Bladder Irrigation (p 235) |
| Nasal Irrigation (p 2027) | Pessaries (p 2341) |
| Throat Irrigation (p 2027) | Vaginal Douche (p 206) |
| | Aerosolization (p 2041) |

Should be referred to specialist

CHAPTER 174

TREATMENT BY INJECTION OR ASPIRATION

INJECTION and aspiration of enclosed body cavities and tissues are performed by means of the hollow steel needle. Most of the techniques are relatively safe and simple and can be performed by the practitioner or by his office assistant; others, however, are procedures for the specialist. Some of them are more conveniently described with the clinical abnormalities of the organs concerned. Page references to all of them are listed below.

| | |
|---|---|
| Paracentesis of Pericardium (p. 802) | Hypodermoclysis (p. 3771) |
| Thoracentesis (p. 2030) | Intramuscular Injection (p. 3772) |
| Artificial Pneumothorax (p. 2033) | Intravenous Injection by Syringe (p. 3773) |
| Abdominal Paracentesis (p. 1920) | Intravenous Infusion by Drip Method (p. 3775) |
| Abdominal Puncture* (p. 1823) | Plasma Infusion (p. 3778) |
| Pneumoperitoneum* (p. 2611) | Intramedullary Infusion and Transfusion (p. 3778) |
| Spinal Anesthesia (p. 3922) | Indirect Blood Transfusion (p. 378) |
| Caudal Anesthesia (p. 3922) | Intracardiac Injection (p. 3779) |
| Peripheral Nerve Block (p. 3919) | Phlebotomy (p. 3780) |
| Paravertebral Nerve Block* (p. 3919) | Spinal Puncture (p. 3782) |
| Joint Aspiration* (p. 3949) | Intraspinal Injection (p. 3782) |
| Lavage of Bursa* (p. 2001) | Cisternal Puncture (p. 3783) |
| Injection of Sclerosing Solutions for Hemorrhoids and Varicose Vein (p. 3941) | Ventricular Puncture (p. 3783) |
| Intradermal Injection (p. 3770) | |
| Subcutaneous Injection (p. 3770) | |

INTRADERMAL INJECTION

Equipment

- Tuberculin syringe
- $\frac{3}{8}$ inch 26 gauge needle

Technic

1. Place skin of forearm on stretch. Cleanse with alcohol.
2. Insert needle obliquely through layers of epidermis.
3. Inject fluid slowly so as to raise a wheal about the diameter of a dime.

Indications

1. Desensitization (p. 563)
2. Immunization (p. 79)

SUBCUTANEOUS OR HYPODERMIC INJECTION

Equipment

- Syringe (2 cc.)
- $\frac{1}{2}$ inch 26 gauge needle

* Should be referred to proper specialist.

Technic

- 1 If injecting contents of vial draw material into sterilized syringe and expel air
- 2 If tablet is to be used dissolve in boiling water in spoon, draw into syringe expel air and cool before injection
- 3 Clean skin with alcohol and hold taut by stretching a segment between thumb and index finger of free hand
- 4 Plunge needle boldly into skin at 45 degree angle inject quickly
- 5 Withdraw needle and massage gently

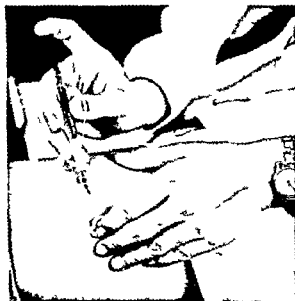


Fig 1104—Injection of insulin After the needle has been inserted the fold of skin is released The plunger is then pushed home

HYPODERMOCLYSIS

Equipment

- Gravity flask
- Single length of rubber tubing from flask to Y tube
- Glass Y tube
- Double length of rubber tubing from Y tube to needles
- Connecting pieces to fit 3 inch 16 gauge needles
- Normal saline 5 per cent dextrose in saline or Ringer's solution
- Hypodermic syringe with 2 per cent procaine

Technic

- 1 Sterilize skin of anteromesial aspects of thighs or axillae
- 2 Make intradermal injections of 2 per cent procaine at sites for clysis
- 3 Assemble clysis set and fill with fluid expelling air from entire apparatus

Wilder Clinical Diabetes Mellitus and Hyperinsulinism

- 4 After five minutes insert clysis needles full length through anesthetized areas avoid entering muscle or breast tissue
- 5 Connect needles to clysis set and start flow regulating rate to avoid painful tension

Indications

Hypodermoclysis is rapidly becoming an obsolete procedure due to the perfection of the intravenous drip. Its chief utility persists in the parenteral administration of fluids to infants and obese patients whose veins are difficult to enter.

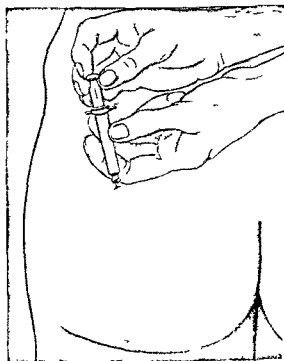


Fig 110c—Aspiration to test for the presence of the needle in a vein

INTRAMUSCULAR INJECTION

Equipment

- 2 cc syringe
- 20 gauge $1\frac{1}{2}$ inch needle
- 26 gauge $1\frac{1}{2}$ inch needle

Technic for Irritants such as Bismuth and Mercury

- 1 Patient lies prone with toes together and heels separated
- 2 Cleanse inner angle of outer upper quadrant of buttock
- 3 Plunge detached needle deep into tissue
- 4 If there is no blood flow attach syringe containing fluid and 0.5 cc of air
- 5 Inject fluid and air

- 6 Pinch area of injection with free hand and quickly withdraw needle

* * * *

Technic for Non irritants such as Androgen and Lstrogon in Oil

- 1 Patient sits on chair or table with legs hanging
- 2 Pull up oily solution into syringe using 18 gauge needle

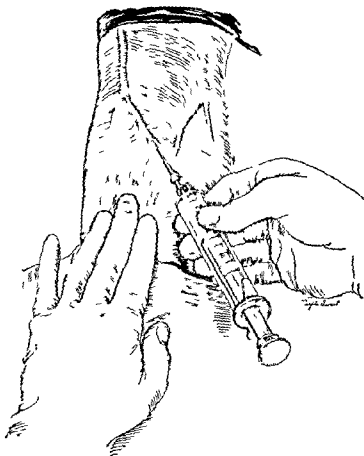


Fig. 1106—Syringe technic. First motion entering the skin showing the tension exerted by the left hand to fix the vein and the pressure with the right for the purpose of steadying the syringe. The needle point is visible under the skin directly over the vein.

- 3 Replace large needle with 26 gauge needle make injection into anterior thigh muscles in median line midway between knee and groin

INTRAVENOUS INJECTION BY SYRINGE

Equipment

2 cc 5 cc or 20 cc syringe

Stokes Beerman and Ingraham Modern Clinical Syphilology

- $\frac{1}{2}$ or 1 inch 24 gauge needle or
1 or $1\frac{1}{2}$ inch 18 gauge needle

Technic

- 1 Inflate blood pressure cuff midway between systolic and diastolic pressures
- 2 Choose vein by palpation select one that is deep and fixed avoid superficial movable vessels if possible
- 3 If biological is to be injected prepare second syringe with 1 cc of $\frac{1}{1000}$ epinephrine
- 4 Clean skin and place skin on stretch with free hand
- 5 Slowly introduce needle through skin parallel to long axis of vein about $\frac{1}{2}$ inch below site of intended puncture use 24 26 gauge needle if possible
- 6 Pierce wall of vein and verify position of needle by aspiration of blood into barrel
- 7 Deflate cuff and relax skin tension
- 8 Slowly inject fluid
- 9 When contents have been expelled aspirate 1 cc of blood
- 10 Apply pressure to site of puncture withdraw needle and have patient hold arm upright for 2 minutes to avoid hematoma

Reactions Local and systemic reactions may be caused by intravenous injections. The former are the result of technical difficulties and arise from perivenous infiltrations often due to piercing of the deeper wall of the vein. The latter may be specific allergic or nonspecific.

Reactions—The *pharmacologically specific* reaction depends upon the nature of the drug employed. Thus the intravenous injection of epinephrine by causing a sharp elevation in the blood pressure may produce a pounding headache palpitation a sensation of constriction in the chest cardiac irregularity tremor flushing or pallor. An injection of a vasodilator such as nitroglycerine may cause intense flushing and a pounding headache faintness or even syncope due to the rapid descent in pressure.

Allergic (anaphylactic) shock may accompany the injection of a biological. In patients who give a history of sensitivity such as vasomotor rhinitis hay fever or bronchial asthma the injection of a protein molecule is preceded by skin or conjunctival testing (p 558). The intravenous injection of a biological is not done unless there is at hand a second hypodermic syringe containing *epinephrine*.

The third or *nonspecific reaction* that follows immediately upon an intravenous injection is in all likelihood a technical error resulting from the rapidity of administration. This syndrome called *speed shock* has been variously termed *anaphylactoid* *nitritoid* or *hemoclastic crisis* *peptone shock* and acute dilatation of the heart. The symptoms closely simulate those of true anaphylactic shock in that the patient becomes flushed and uncomfortable and complains of a pounding headache or constriction of the chest. There may be a respiratory or pulse irregularity. In extreme instances there is cessation of respiration or disappearance of the radial pulse. Deaths have been reported. The best treatment of *speed shock* is prevention by *slow injection*.

INTRAVENOUS INFUSION BY THE DRIP METHOD

Equipment

Infusion reservoir

Solutions (Commercial sets available council approved)

Physiological saline

5 10 20 and 25 per cent dextrose in distilled water

2 5 7.5 10 20 and 25 per cent dextrose in saline

3 and 5 per cent sodium chloride

Ringer's solution

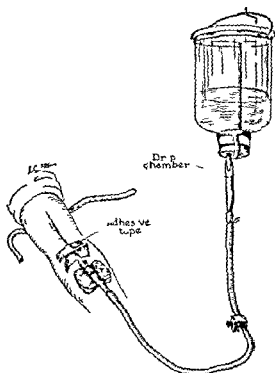


Fig 1107—Intravenous administration of fluid

5 and 10 per cent dextrose in Hartmann's solution

M/6 Sodium- γ lactate

Lactate Ringer's solution (Hartmann)

5 and 10 per cent dextrose in Hartmann's solution

Rubber tubing 30 cm in length from reservoir to Murphy drip

Hoffman clamp for regulation of rate of flow

Rubber tubing 150 cm in length from Murphy drip to needle

connecting piece to fit 18 gauge $1\frac{1}{2}$ inch needle

2 cc syringe

Christopher Major Surgery

Technic

- 1 Assemble set and expel all air from apparatus
- 2 Perform vein puncture as above choose vein of forearm if possible avoid antecubital vessel unless other veins cannot be managed.
- 3 Slowly insinuate needle full length tape in place with adhesive be sure air is expelled from rubber tubing connect needle to infusion set open Hoffman clamp and begin flow
- 4 Regulate rate of flow by Hoffman clamp to 30-60 drops per minute
- 5 Protect skin under needle by inserting vaseline gauze cover with sterile gauze
- 6 Immobilize arm with posterior splint concave crib or folded pillow
- 7 Avoid pressure posteriorly on musculo spiral nerve
- 8 Temperature regulation unnecessary except for cooling with penicillin
- 9 If drip is to be continued give mouth wash and oral hygiene three times daily to prevent parotitis (p 4015)

Technical Difficulties—*Cessation of flow* in the drip may result from pressure on the rubber tubing or kinking from some external pressure If none of these simple deterrents is operative the needle is likely to be at fault The position of the needle can be changed by rotation A stylet may be inserted to dislodge a small clot It should rarely be necessary to remove the drip apparatus if it has been freely running for any length of time

In *children* and occasionally in adults it may be impossible to enter a superficial vein Under these circumstances it is necessary to cut down on the vessel A transverse incision is made over a palpable vein If none can be located the antecubital fossa is explored A tourniquet is applied so as to distend the vessel Two ligatures are passed beneath the vein and the distal ligature is tied The vein is incised about 2 cm above the tied ligature A large cannula is introduced into the vein and secured proximally with the untied ligature and distally with the tied ligature The skin is temporarily closed over the cannula with a single suture The wound is protected with a layer of sterile petrolatum The connecting tip from the drip set is set into the cannula and the infusion is instituted as previously described Ankle veins may be employed if it is inconvenient to use those in the antecubital fossa In *infants* before the fontanelles have closed intravenous injections may be given into the longitudinal sinus readily entered mesially in the anterior fontanelle

Addition of Drugs and Sera—If it becomes necessary to give drugs parenterally during the course of an intravenous infusion they may be placed directly in the reservoir or injected into the rubber tubing This is the equivalent of an intravenous introduction Through the slow intravenous drip sera of various types may be introduced in relatively enormous quantities

Reactions—Reactions from the intravenous drip other than local phlebitis are rare Occasionally due to the presence of pyrogenic bacteria in inadequately prepared solutions *chills* may occur (p 32) Usually some cause other than the intravenous drip is responsible for such chills If there is doubt the drip should be discontinued

Anaphylactic responses are prevented in the experimental animal by the slow drip. Even the sensitized animal will not develop shock if the rate of flow is reduced. This should not deter the clinician from applying all safeguards such as *skin testing desensitization* where necessary and keeping available an *epinephrine solution*.

Bull reactions are not to be feared with a normal circulation. The circulatory system readily handles the increased mass of fluid.

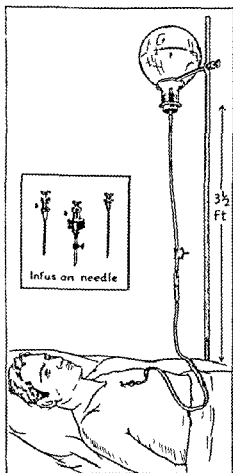


Fig. 1108.—Infusions of fluids through the marrow of the manubrium.

unless it is on the verge of failure. Cardiac patients on the edge of compensation may develop constriction of the chest and peripheral and pulmonary edema if a daily fluid intake from all sources exceeds 3000 to 4000 cc.

The fear of causing or increasing bleeding is unwarranted. Following hemoptysis or hematemesis many physicians hesitate to employ a drip lest bleeding be resumed or perpetuated. This dread is not warranted.

Courtesy of Dr. L. M. Tocantins.

experimentally or practically since blood pressure levels are not elevated above normal by the intravenous infusion. A shock level may be raised to normal. The normal level is not exceeded.

PLASMA INFUSION

Equipment

- As for intravenous drip
- Lyophile plasma
- Ampoule of sterile distilled water

Technic

- 1 Dilute the lyophile plasma with equal parts of sterile distilled water ordinarily 250 cc of each are employed
- 2 Follow manufacturer's directions to assemble special apparatus
- 3 Begin infusion of plasma but increase rate of flow if patient is in shock (p 928)

INTRAMEDULLARY INFUSION AND TRANSFUSION

Equipment

- As for intravenous drip
- 19 gauge $\frac{1}{2}$ inch needle with stilet

Technic

- 1 Insert needle into medullary cavity as for sternal puncture
- 2 Infuse solution or blood by intravenous drip technic

Indications

- 1 In marked hypersensitivity
- 2 When veins are inaccessible
- 3 In treatment of leukemia (p 1100) and myelophthisic anemia (p 1091)

INDIRECT TRANSFUSION

Equipment

- Phlebotomy needle
- Collecting cylinder of 500 cc capacity (vacuum sealed)
- Sterling stirring rod
- Ampoule of 50 per cent sodium citrate (5 cc)
- Intravenous drip set

Technic

- 1 Collect blood from recipient and prospective donors
- 2 After grouping cross match and determine Rh status of donor and recipient (p 3779)
- 3 Get Kahn test on donor spread for malaria (p 507) and blood count do physical examination with special attention to evidences of primary syphilis (p 3278)
- 4 If tests for syphilis are negative blood count is satisfactory physical examination is normal and cross matching reveals neither agglutination nor hemolysis donor reports after 6 hours fast
- 5 Collect 500 cc of blood in cylinder to which 5 cc of 30 per cent sodium citrate had previously been added stir constantly filter through sterile gauze if necessary

- 6 Set up intravenous drip (p 3775) add blood to reservoir and transfuse

Reactions—The transfusion of whole blood may give rise to the reactions and technical difficulties inherent in any intravenous drip (p 3776). Additionally there may be specific disturbances of various types *urticarial eruptions* are frequent and are usually due to foreign protein recently ingested by the donor *chills* occur in 1 to 2 per cent of the instances under best conditions and are due to pyrogenic contaminants accidentally introduced. Much more serious are the accidents due to errors in blood grouping or matching (p 3710).

Three types of hemolytic transfusion reactions may occur. All give rise to hemolysis, hemoglobinemia, hemoglobinuria, hematuria, anuria and jaundice with an occasional fatality.

The simplest type of hemolytic transfusion reaction is due to an error in technic in which distilled water has been used instead of normal saline solution. A second cause is the incorrect grouping by a laboratory of patient or donor (p 3710). The third type of hemolytic transfusion reaction results from intra group incompatibility relative to the Rh factor described more fully under erythroblastosis foetalis (p 2761).

The Rh factor is present in the red blood cells of approximately 85 per cent of the population. Individuals whose red cells possess the factor are called Rh positive while those in whom the factor is absent are known as Rh negative. The Rh factor has no relationship to the major blood groups.

Rh factors may be inherited or acquired. In the latter instance the Rh negative individual becomes immunized by Rh positive blood as the result of previous transfusion or of childbirth in which the fetus is Rh positive. To prevent the occurrence of intra group hemolytic transfusion reactions it is necessary to determine the Rh grouping of patients who have received transfusions in the past, those who are to receive frequent transfusions and women in the latter part of pregnancy. Rh positive patients may be transfused with either Rh positive or Rh negative blood provided that there is no major blood group incompatibility. Patients with Rh negative blood are best transfused with Rh negative blood if it is at all possible. Under unusual circumstances if only Rh positive blood is available for the Rh negative recipient a biologic test is performed by making a probatory transfusion of 50 cc. of Rh positive blood. If any clinical reaction or elevation of the icterus content of the blood is noted, larger amounts should not be given. However, if the probatory transfusion is without event the remainder of the blood may be given after two hours.

INTRACARDIAC INJECTION

Equipment

- 2 cc. syringe
- 20 gauge 4 inch needle
- Epinephrine $\frac{1}{1000}$

Technic

- 1 Reserve injection for recent cardiac arrest
- 2 Insert needle along right border of sternum in third or fourth interspace until blood is aspirated

experimentally or practically since blood pressure levels are not elevated above normal by the intravenous infusion. A shock level may be raised to normal. The normal level is not exceeded.

PLASMA INFUSION

Equipment

- As for intravenous drip
- Lyophile plasma
- Ampoule of sterile distilled water

Technic

- 1 Dilute the lyophile plasma with equal parts of sterile distilled water ordinarily 250 cc of each are employed
- 2 Follow manufacturer's directions to assemble special apparatus
- 3 Begin infusion of plasma but increase rate of flow if patient is in shock (p 928)

INTRAMEDULLARY INFUSION AND TRANSFUSION

Equipment

- As for intravenous drip
- 19 gauge $\frac{1}{2}$ inch needle with stilet

Technic

- 1 Insert needle into medullary cavity as for sternal puncture
- 2 Infuse solution or blood by intravenous drip technic

Indications

- 1 In marked hypersensitivity
- 2 When veins are inaccessible
- 3 In treatment of leukemia (p 1100) and myelophthisic anemia (p 1091)

INDIRECT TRANSFUSION

Equipment

- Phlebotomy needle
- Collecting cylinder of 500 cc capacity (vacuum sealed)
- Sterling stirring rod
- Ampoule of 50 per cent sodium citrate (5 cc)
- Intravenous drip set

Technic

- 1 Collect blood from recipient and prospective donors
- 2 After grouping cross match and determine Rh status of donor and recipient (p 3779)
- 3 Get Kahn test on donor spread for malaria (p 507) and blood count do physical examination with special attention to evidences of primary syphilis (p 3278)
- 4 If tests for syphilis are negative blood count is satisfactory physical examination is normal and cross matching reveals neither agglutination nor hemolysis donor reports after 6 hours fast
- 5 Collect 500 cc of blood in cylinder to which 5 cc of 30 per cent sodium citrate had previously been added stir constantly filter through sterile gauze if necessary

- 6 Set up intravenous drip (p 3775) add blood to reservoir and transfuse

Reactions—The transfusion of whole blood may give rise to the reactions and technical difficulties inherent in any intravenous drip (p 3770). Additionally there may be specific disturbances of various types *urticarial eruptions* are frequent and are usually due to foreign protein recently ingested by the donor *chills* occur in 1 to 2 per cent of the instances under best conditions and are due to pyrogenic contaminants accidentally introduced. Much more serious are the accidents due to errors in blood grouping or matching (p 3710).

Three types of hemolytic transfusion reactions may occur. All give rise to *hemolysis hemoglobinemia hemoglobinuria hematuria anuria* and jaundice with an occasional fatality.

The simplest type of hemolytic transfusion reaction is due to an error in technic in which distilled water has been used instead of normal saline solution. A second cause is the incorrect grouping by a laboratory of patient or donor (p 3710). The third type of hemolytic transfusion reaction results from intra group incompatibility relative to the Rh factor described more fully under *erythroblastosis foetalis* (p 2761).

The Rh factor is present in the red blood cells of approximately 85 per cent of the population. Individuals whose red cells possess the factor are called Rh positive while those in whom the factor is absent are known as Rh negative. The Rh factor has no relationship to the major blood groups.

Rh factors may be inherited or acquired. In the latter instance the Rh negative individual becomes immunized by Rh positive blood as the result of previous transfusion or of childbirth in which the fetus is Rh positive. To prevent the occurrence of intra group hemolytic transfusion reactions it is necessary to determine the Rh grouping of patients who have received transfusions in the past; those who are to receive frequent transfusions and women in the latter part of pregnancy. Rh positive patients may be transfused with either Rh positive or Rh negative blood provided that there is no major blood group incompatibility. Patients with Rh negative blood are best transfused with Rh negative blood if it is at all possible. Under unusual circumstances if only Rh positive blood is available for the Rh negative recipient a biologic test is performed by making a probatory transfusion of 50 cc. of Rh positive blood. If any clinical reaction or elevation of the icterus content of the blood is noted larger amounts should not be given. However if the probatory transfusion is without event the remainder of the blood may be given after two hours.

INTRACARDIAC INJECTION

Equipment

- 2 cc syringe
- 20 gauge 4 inch needle
- Epinephrine $\frac{1}{1000}$

Technic

- 1 Reserve injection for recent cardiac arrest
- 2 Insert needle along right border of sternum in third or fourth interspace until blood is aspirated

- 3 Slowly inject epinephrine, aspirate and reinject blood for mechanical stimulation

PHLEBOTOMY

Equipment

- 1 Special needle 13 15 gauge 2 inches long with obturator and stilet
- 2 Collecting cylinder preferably of vacuum type
- 3 2 cc syringe with 2 per cent procaine

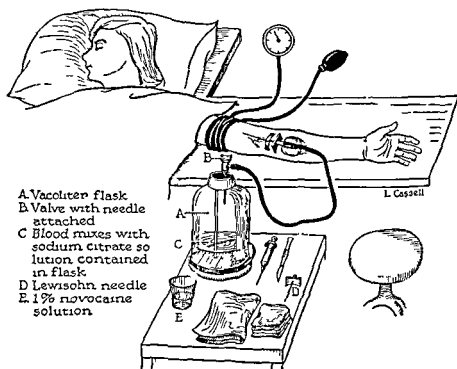


Fig 1109 —Commercial vacoliter flasks such as the one shown in this illustration have greatly simplified the withdrawal of blood from a donor. The rubber stopper is covered with an air tight rubber disc to preserve the vacuum. The special needle which is attached to the valve mechanism is passed through this rubber disc after being connected with the rubber tubing. After the needle is inserted into the donor's arm the valve is carefully opened and regulated so as to produce a brisk steady flow of blood. When the desired amount of blood has been taken the valve needle is withdrawn the air tight rubber disc is peeled off and the tube to the recipient (which contains the metal filter) is connected with the flask by the insertion of a glass tip in the rubber stopper. The flask is then inverted and suspended from a standard at a height to insure flow by gravity.*

Technic

- 1 Inflate blood pressure cuff midway between systolic and diastolic levels
- 2 Produce intracutaneous wheal of procaine at site for introduction of phlebotomy needle
- 3 Introduce phlebotomy needle into vein
- 4 Withdraw stilet and obturator

*Jennings in Christopher Textbook of Surgery

- 5 Collect blood in cylinder to which 5 cc of 30 per cent sodium citrate have been added if blood is to be used for transfusion (p 3778)
- 6 Deflate cuff withdraw needle apply firm pressure

SPINAL PUNCTURE

Equipment

- Special lumbar puncture needles with obturators (19 to 20 gauge 3-3½ inches long)
- 2 cc syringe with procaine
- Sterile test tubes
- Water manometer (Quincke)



Fig 1110—Introducing the needle between the two fingers of the left hand with the right hand resting upon the left wrist as a guide and a guard against sudden movements or excessive pressure which may break the needle

Technic

- 1 Give preliminary dose of phenobarbital 0.090 gm (1½ grains) orally or dilaudid 0.002 gm (½ grain) subcutaneously
- 2 Position patient with hips and shoulders at edge of table jack knife in lateral decubitus with assistant holding head and knees in maximum flexion
- 3 Mark injection site at level of L 3 and 4 or L 4 and 5 (line joining iliac crests) introduce procaine with 2 cc syringe
- 4 With thumb of free hand braced on lower spinous process introduce lumbar puncture needle in median line directed slightly upward
- 5 At a distance of 1½ or 2½ inches resistance decreases as canal is entered
- 6 Withdraw obturator partially and collect fluid drop by drop

- 3 Slowly inject epinephrine aspirate and reinject blood for mechanical stimulation

PHLEBOTOMY

Equipment

- 1 Special needle 13 15 gauge 2 inches long with obturator and stilet
- 2 Collecting cylinder preferably of vacuum type
- 3 2 cc syringe with 2 per cent procaine

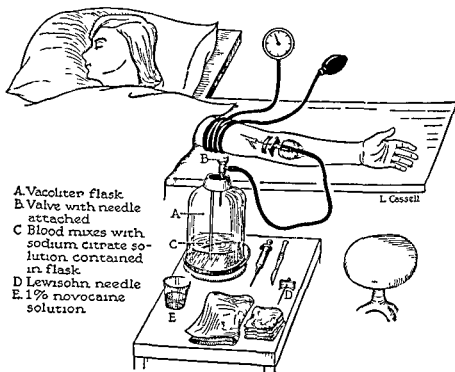


Fig 1109.—Commercial vacoliter flasks such as the one shown in this illustration have greatly simplified the withdrawal of blood from a donor. The rubber stopper is covered with an air tight rubber disc to preserve the vacuum. The special needle which is attached to the valve mechanism is passed through this rubber disc after being connected with the rubber tubing. After the needle is inserted into the donor's arm the valve is carefully opened and regulated so as to produce a brisk steady flow of blood. When the desired amount of blood has been taken the valve needle is withdrawn the air tight rubber disc is peeled off and the tube to the recipient (which contains the metal filter) is connected with the flask by the insertion of a glass tip in the rubber stopper. The flask is then inverted and suspended from a standard at a height to insure flow by gravity.

Technic

- 1 Inflate blood pressure cuff midway between systolic and diastolic levels
- 2 Produce intracutaneous wheel of procaine at site for introduction of phlebotomy needle
- 3 Introduce phlebotomy needle into vein
- 4 Withdraw stilet and obturator

- 4 For spinal anesthesia collect spinal fluid in ampoule containing sterile procaine crystals when solution has occurred reintroduce fluid slowly with syringe

CISTERNAL PUNCTURE

Equipment

- Spiegel needle (19 gauge $2\frac{2}{3}$ or $3\frac{1}{8}$ inches long with guard at 1 or 1.5 inches and obturator)

Technic

- 1 Shave hair below occiput
- 2 Introduce needle in median line along an imaginary line joining glabella and external auditory meatus

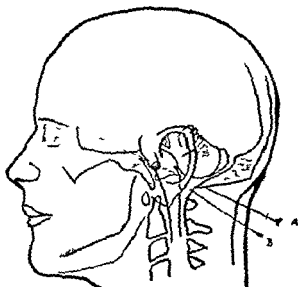


Fig. 111.—Sagittal section of head showing anatomical relations involved in cisternal puncture

- 3 At about 3 cm note give after piercing atlanto-occipital ligament Do not insert needle beyond guard
- 4 Remove obturator and collect spinal fluid as above

VENTRICULAR PUNCTURE

Equipment

- Fontanelle needle (20 gauge $\frac{5}{8}$ inch)

Technic

- 1 Before fontanelle has closed in infancy enter ventricle at lateral angle of anterior fontanelle
- 2 At distance of 3-5 cm withdraw spinal fluid

Courtesy of Dr. Leo Spiegel

- 7 If there is no flow rotate needle
- 8 This maneuver failing replace obturator and gently insert needle further
- 9 If flow is not obtained or bone is palpated by needle redirect position until canal is entered the dry tap is a great rarity
- 10 After collecting a few drops of fluid in first tube let a few more drops flow in second tube and the remainder in the third tube Use second tube for cytology and chemistry (p 3735) use third tube for culture and serology (p 3736) do not remove more than 8-10 cc
- 11 Withdraw needle apply dry dressing place patient on back with out pillow and advise immobilization for several hours

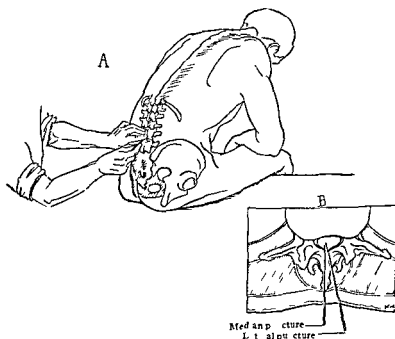


Fig 1111—Technic of lumbar puncture *A* The left forefinger locates the area between the fourth and fifth lumbar spinous processes *B* is a cross section at this level to show the median and lateral insertion of the needle into the subarachnoid space

INTRASPINAL INJECTION AND SPINAL ANESTHESIA

Equipment

- As for lumbar puncture
- Vial of sterile medicinal

Technic

- 1 Perform lumbar puncture as above
- 2 For intraspinal injection of serum sulfonamide or penicillin with draw 1-2 cc more than amount of planned injection
- 3 Connect needle to gravity reservoir and infuse medicinal using 1-2 cc less than amount of spinal fluid withdrawn

PHYSICAL THERAPY BY THE USE OF COLD

Local Cryotherapy—The Sparklet bulbs are placed in a metal expansion chamber in which the carbon dioxide is converted into snow and moulded and compressed into a solid capsule about $\frac{5}{16}$ inch in diameter and 2 inches long. Local application of the snow pencil damages local vessels with a subsequent exudation of serum and the formation of a bulla. The blood supply to the frozen area is reduced; there is a resultant death of tissue whose extent depends on duration of contact and the degree of pressure that has been applied.

TABLE 201—THE USE OF COLD IN PHYSICAL THERAPY

| Method | Technic | Remarks |
|---|---|---|
| Local application | Ice bag, ice collar, ice coil, cold compresses | To contract local vessels, diminish local blood flow and exudation in acute inflammations, hemorrhage and injuries |
| Systemic application | Cold sponge, cold or ice pack | To reduce temperature in hyperpyrexia or insolation for sedative effect on febrile patients |
| Cold water | Place hand in basin of cold water for 90 seconds | See Essential Hypertension (p. 300). Blood pressure rise of more than 20 mm. |
| Cold shower | | Tonic and stimulant |
| Freezing with ethyl chloride (p. 3715) | Local spray | Local anesthesia (p. 4914) |
| Local cryotherapy | CO ₂ snow made in Sparklet bulb approved by the Council on Physical Medicine of the A.M.A. | For removal of warts, corns, lichen, hemus, keratoses, angiomas, xanthomas, nevi and moles; apply with 1 pound pressure for 10-60 seconds |
| Local cryotherapy in amputations (p. 3785) | Refrigeration and application of tourniquet | Provides anesthesia, prevents bleeding and shock and reduces mortality |
| Systemic cryotherapy (refrigeration bulb in motion) | Reduce body temperature to 80-84 F. | To arrest tissue growth in leukemia (p. 1100) and inoperable malignancy (p. 577) |

Under ordinary circumstances the treated area appears hard and depressed immediately following the application of the pencil. Within a few moments the tissues resume normal color and contour. Approximately thirty minutes later a circumscribed wheal develops which later forms into a vesicle. The latter should be preserved as a protective dressing, though its content may be drained off by a sterile needle if there is local discomfort. After a few days a firm crust is formed; this remains until the underlying tissue is completely healed.

Local Cryotherapy in Amputations—Refrigeration anesthesia for amputation of an extremity is carried out as follows:

- 1 The extremity is elevated to drain blood.
- 2 An Esmarch bandage is applied unless there is infection.

CHAPTER 175

PHYSICAL THERAPY

THE various methods of physical therapy are

Exercise (p 3757)

Massage (p 3766)

Occupational Therapy (p 3760)

Climatotherapy (p 3761)

Balneotherapy (p 3763)

Spa Therapy (p 3764)

Appliances and Braces (p 3070)

Cold and Cryotherapy (p 3 83)

Heat and Hyperthermy (p 3786)

Baths (Hydrotherapy) (p 3792)

Electrotherapy (p 3792)

Radiant Energy including Heliotherap

Roentgen Therapy and Radium Ther
apy (p 3796)

Physical therapy is almost universal in its practice. It probably antedates all other forms of medical treatment and had already reached a state of high development in the Roman baths at a time when many other forms of therapeutics were not even known.

Physiotherapy may be practiced in the home, in the physician's office, in the more fully equipped establishment of the physiotherapist or at the highly developed physiotherapeutic institute. Its technique may be accomplished by the patient, the practitioner or the specialist. Of all the methods of treatment, it is the most capable of exploitation by charlatans, many of whom abuse public faith by overenthusiastic and unfulfilled claims.

Physiotherapy in the Home—With the cooperation of his patient, the ingenious practitioner may be able to convert the modern household into a remarkably complete physiotherapeutic institute. Without great trouble or expense, the patient can provide himself with heat, cold, infra red and ultraviolet rays and air conditioning. The modern bathroom is equipped for hydrotherapy by the shower or the various baths (p 3791). Electrotherapy can be employed by means of the electric pad or the infra red or ultraviolet rays; the modern refrigerator supplies the necessities for cryotherapy.

Office Physiotherapy—In his office, the practitioner supplements the domestic physiotherapeutic institute with slightly more elaborate equipment, such as a diathermy machine by which he can give heat treatments, electrodesiccation and electrocoagulation. At negligible expense, he may purchase kits for local cryotherapy and iontophoresis.

The Physiotherapist—The services of the physiotherapist are required for the operation of the more complicated forms of apparatus and those in which there is a treatment hazard. Refrigeration, systemic cryotherapy, hyperthermy, underwater and corrective exercise, faradism, galvanism, sinusoidal currents, electrolysis, the more extensive use of electrocoagulation, surgical endothermy, electroconvulsions, roentgen therapy and the application of radium are each and all in the province of the consultant.

Despite the complexity of many of the physiotherapeutic procedures, the overwhelming majority of indications can be met either in the home or in the practitioner's office. These are summarized in Table 01 and 20.

PHYSICAL THERAPY BY THE USE OF HEAT

Prevention of Heat Loss.—Increase in local heat is most simply accomplished by the prevention of heat loss through tight wrapping with a nonporous material. Examples of this are the efficacy of the *flannel binder* for the chest or the abdomen, the familiar red flannel wrapped about the neck in tonsillitis, or the use of the popular sweatshirt.

Application of Dry Heat.—The application of the *hot brick*, the *water bag* or the *electric pad* is simple and effective. In the use of these modalities the patient is warned concerning burns. A layer of flannel is interposed between the source of heat and the skin. Heat is applied cautiously, if at all, to the patient who has local anesthesia or analgesia. *The electric pad is removed during sleep. It should not be employed with wet dressings or compresses lest a short circuit occur.*

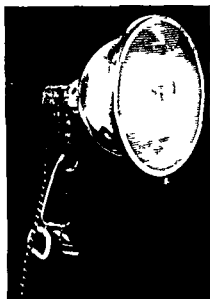


Fig. 1113.—A nonexpensive luminous heat lamp with clamp attachment.

Infra red Rays.—The infra red generators are relatively inexpensive. They are of two types—the spiral coil and the plate or disk models. When current is passed through these generators they become hot and emanate infra red rays of a spectral emission varying between 2000 and 5000 millimicrons. Infra red irradiations do not penetrate the skin. They are merely heat rays. The source of infra red is placed a comfortable distance from the patient. It is employed for as long as desirable. Burns should not occur unless there is local analgesia or local circulatory deficiencies.

Luminous Heat.—Luminous heaters are improvised by using a 250 watt Mazda C X lamp or four 60 watt tungsten filament bulbs housed in a cup shaped reflector made of polished steel metal to resemble the popular cradle baker employed in institutional work. The use of these cradles

- 3 The leg is placed on rubber sheeting and cracked ice is packed to the level where the tourniquet will later be applied
- 4 The head of the bed is raised to assist drainage of the melted ice.
- 5 After 30 minutes the tourniquet is applied 3-6 inches above incision level, the skin temperature is kept at 40° F

TABLE 202—THE USE OF HEAT IN PHYSICAL THERAPY

| Method | Technic | Remarks |
|-------------------------------------|--|---|
| Prevention of heat loss by wrapping | Flannel binder sweatshirt | Increase local blood flow produce local vasodilatation relieve local pain produce local diaphoresis and increase local processes of immunity (?) in inflammations and injuries |
| Application of dry heat | Hot brick hot water bottle electric pad | |
| Infra red rays | Inexpensive generators emanate rays of spectral emissions of 2000-5000 millimicrons no penetration no burn | |
| Luminous heat | 250 watt Mazda C lamp in cup-shaped cradle reflector of polished steel metal | |
| Moist heat applied locally | Hot or warm compresses wet dressings poultice of flaxseed or mustard fomentations stupes arm baths foot baths sitz baths throat irrigations enemas vaginal douches | |
| Local diathermy | By high frequency long wave (500 000 to 3 000 000 cycles per second) or short wave (10 000 000 to 100 000 000 cycles per second) | Local physiologic effects limited to the effects the heat produces no demonstrable selective thermal action no action on cells or tissues except through heat produced (Council on Physical Medicine) |
| Systemic heat | Hot baths hot packs | Diaphoresis in poisoning |
| Hyperthermy | By therapeutic malaria (p 3779) intravenous typhoid vaccine (p 1880) intramuscular boiled milk (p 1379) or hyperthermia (p 3789) | In early syphilis (p 3218) and gonorrhea (p 217) in paresis and tabes (p 1379) for chorea (p 1417) and brucellosis (p 314) may be combined with chemotherapy (p 340) |

- 6 Amputation is performed after an interval of 2½ hours for low or mid thigh 2 hours for upper third of leg 1½ hours for mid leg and 1 hour for metatarsal level
- 7 After operation the stump is placed in a posterior moulded splint and ice is reapplied for 48-72 hours

Systemic Cryotherapy—Systemic cryotherapy hailed as a possible palliative measure for the control of inoperable cancer (p 577) seems not to have fulfilled its promise though reliable investigators have reported some success in the relief of intractable pain

thermy machine should choose one that has the approval of the Council on Physical Medicine of the American Medical Association. The manufacturer's catalogue gives full directions as to the installation and operation of the machine which can be used also for *electrodesiccation*, *electrocoagulation* and *endothermy* (p 3792).

Special attention is directed to the precautions that must be exercised in any type of diathermy. Burns and electric shock constitute hazards that can be prevented by removal of all metallic objects from the area of treatment, removal of clothes from the parts under treatment, the use

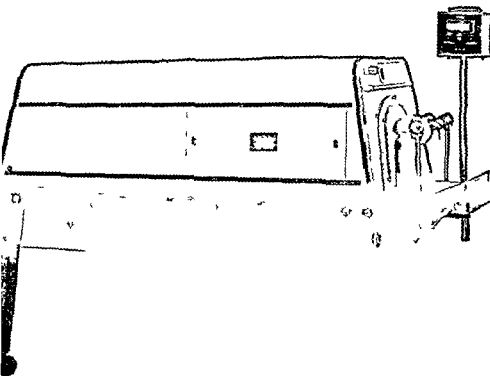


Fig 1115—The hyper therm (close t)

of a chair or table that is free from metal parts, avoidance of uncomfortable heat and pain, and refusal to treat an area in which there is anesthesia or diminution in local blood supply.

Hyperthermy—The introduction of hyperthermy or artificial fever treatment is one of the outstanding contributions of the twentieth century. The practitioner who is dependent upon his own resources may utilize intramuscular injections of boiled milk, intravenous injections of typhoid vaccine, or the production of a therapeutic malaria (p 1379). Hyperthermy may also be accomplished without the purchase of elaborate

is similar to that of the infra red lamp Either constitutes a convenient, satisfactory and safe source of thermal therapy

Diathermy—The tempered opinion of the Council on Physical Medicine of the American Medical Association is not held by many of the enthusiasts for *diathermy* The latter claim a special selective thermal action deeper penetration of tissue specific effects that are independent of heat and specific biological and bactericidal activities They regard short wave diathermy as excelling long wave diathermy and they intimate that different results are obtained with different wave lengths

When experts differ the practitioner is faced with a difficult decision as to procedure The senior author after several years of office trial with diathermy concluded that the modality accomplished nothing that could not be attributed to heat he was unable to observe the specific results claimed by the enthusiasts At most symptomatic relief was encountered In many instances the amelioration was more likely the spontaneous course of the affliction influenced by a measure of psychotherapy although an

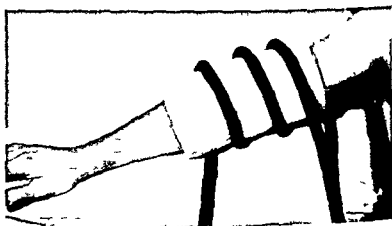


Fig 1114—Method of applying short wave diathermy by means of an induction coil to any section of the arm between the wrist and shoulder*

occasional favorable experience was encountered in a condition that had previously proved obstinate to other forms of therapy Unfortunately there was no consistency in results and no general conclusion could be drawn

Indications for Diathermy—Diathermy is widely employed by enthusiasts for the relief of all skeletal disturbances These include myositis bursitis tenosynovitis fibrositis arthritis sprains strains fractures and all the vague and intangible states that beset the human skeleton Diathermy is also used with enthusiasm in the prevention and treatment of the common colds and other upper respiratory disorders in eye infections in pelvis inflammatory disease in the pyodermas in pneumonia otitis media prostatitis epididymitis and adnexal inflammations Specially devised electrodes are available for introduction into the nose ear vagina and rectum

Technical Considerations—The practitioner who purchases a dia

* Krusen Physical Medicine

TABLE 203—PHYSICAL THERAPY BY MEANS OF BATHS

| Method | Technic | Remarks |
|---------------------------|--|---|
| Hot and cold shower | | Cleansing and tonic |
| Warm tub | At about 100° F | Sedative and cleansing |
| Hot tub | At about 100-108° F may raise body temperature to 104° F combine with warm drink | Hypertermia to abort upper respiratory infections (p. 2114) |
| Continuous bath† | Patient suspended across bathtub on canvas hammock or placed on inner tube at 100° F apply friction anoint hands and feet with oil to prevent maceration | Sedative in acute mania and delirium tremens (p. 1381) |
| Medicinal baths‡ | Line needle oil or epsom salts | Sedative analgesic |
| Colloid baths (p. 3133) | Bran starch | In exudative forms of dermatitis to relieve pruritus (p. 3170) |
| Carbonated bath§ (Naheim) | Artificial production of CO ₂ | In cardiovascular disease |
| Shock bath | Fall of cold water thrown on chest and back while seated in warm tub | In exhaustion states and acute alcoholism (p. 3845) |
| Sitz bath | In tub at 110° F | For anal and pelvic disorders |
| Russian bath | In steam room with massage for 10-20 minutes followed by cold shower or plunge | General tonic effects detoxification (?) recovery from acute alcoholism (p. 3845) |
| Turkish bath | In room of dry heated air at 110-130° F then to room at 150-200° F massage followed by cold shower or plunge | See Russian bath above |
| Calcium baths | Cold compresses about head while body is enclosed in cabinet at 140°-180° F followed by cold shower | Weight reduction |
| Mud bath | Heated mud at 100-125° F for 10-30 minutes | Spa therapy (p. 3764) |
| Paraffin bath | Prevent heat loss by insulating body with melted paraffin | Spa therapy (p. 3764) |
| Whirlpool bath | Hydrotherapeutic institutes | Neuromuscular lesions of extrinsicities |
| Scratch douche | Hydrotherapeutic institutes | Tonic |
| Underwater exercise | Hydrotherapeutic institutes | Muscle reeducation especially in poliomyelitis (p. 2886) |

The hot tub bath in the opinion of the senior author is a useful therapeutic remedy for abatement of the acute upper respiratory infections (p. 2114) since many patients become faint after a few minutes of treatment, some member of the family should be prepared to apply cold compresses to the forehead and assist with dry-g.

† The continuous bath is of nestimal value in the treatment of disturbed patients. It can be improvised in the home by setting the patient on a partially inflated inner tube; needless to say constant attendance is essential.

‡ It is doubtful whether medication adds to the sedative and analgesic effects of the warm tub. Nevertheless there are patients who think more highly of a therapeutic measure if it involves additional expense. of the test manual of a advertising copywriter.

§ The artificial Naheim salt can be purchased at the pharmacy. The directions are to be carefully followed to avoid damage to the bath tub. So far as the senior author has observed, the erosion of the enamel is the only consistently demonstrable effect.

|| The calcium bath is mentioned only to be condemned; the weight loss is due to dehydration which is compensated the next time fluids are ingested.

rate apparatus by surrounding the patient with hot water bottles and blankets as elsewhere described (p 1379)

The most scientific method of producing hyperthermy is the province of the specialist who employs air conditioned cabinets or *hypertherma*. In these the air temperature the relative humidity air velocity and body temperature are capable of accurate control. Under these ideal conditions it is possible to demonstrate that hyperthermy causes an increased heart rate slight fall in blood pressure loss of water and salt through sweating leukocytosis and polynucleosis with a shift to the left a slight alkalosis elevation of the basal metabolic rate and minor changes in the electrocardiogram.

Hyperthermy like all other potent therapeutic measures is not without risk. Patients may succumb to hyperpyrexia collapse azotemia and circulatory failure. This heroic type of therapy should be avoided in patients over the age of 60 and those with demonstrable cardiovascular renal disease tuberculosis undernutrition obesity or abnormalities of liver function.

Application of Moist Heat—Moist heat is applied locally by means of compresses poultices fomentations wet dressings stupes or by the arm or foot bath tubs.

The *hot or warm compress* is prepared by soaking a cloth in hot water wringing it dry and applying the hot cloth to the part. The compress may be used on the head the eye the throat the chest the abdomen or the wound.

The *wet dressing* employs a compress covered by oiled silk to prevent evaporation. It is fixed by a gauze bandage.

The *poultice* is a method of maintaining a more or less constant application of moist heat. One of the oldest and most commonly employed poultices is prepared with flaxseed. Flaxseed meal is stirred in boiling water until the consistency is such that the mixture drops slowly from a spoon. A teaspoonful of olive oil and a pinch of baking soda are added. The poultice is poured evenly onto a sheet of muslin and applied to the oiled skin. The flaxseed retains the heat for a considerable time. Before the days of the hot water bottle or the electric pad it was a popular and useful method of maintaining a constant source of heat.

The *fomentation* differs very little from the compress. It is usually performed by immersing pieces of old blanket in boiling water. The hot blanket is then wrung out dry and applied to skin which has previously been protected by vaseline or olive oil. The fomentations are renewed every 10 or 15 minutes until three or four have been used.

The *stupe* is a fomentation that employs additionally a counterirritant such as rectified spirits of turpentine or camphorated oil.

Moist heat is also provided by soaking the arm or leg in a *tub* or *pail* of hot water. The *arm bath* is of particular value in the treatment of disability or infection of the elbow wrist or hand.

The *foot bath* may be used as a general diaphoretic as in the popular mustard foot bath or for contrast baths in the treatment of the disturbances of the peripheral vessels (p 994). For the contrast baths the feet are plunged alternately into pails of hot and cold water so that the capillary circulation and arterioles are first dilated and then contracted.

PHYSICAL THERAPY BY RADIANT ENERGY

Radiant energy derived from the sun or from artificial sources may be employed for therapeutic effect. The nature of the physiological effect of the ray varies with the wave length which is measured in millimicrons ($\mu\mu$) the equivalent of one one millionth of a millimeter. The wave lengths are also measured in *angstrom* units which equal one ten millionth of a millimeter.

The electromagnetic spectrum passes through infra red rays visible rays ultraviolet rays roentgen rays and gamma rays.

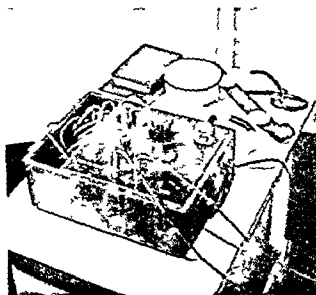


Fig 1116—Electroshock unit. Showing treatment button timer switch fuse on-off switch potentiometer to control voltage neon bulb (indicates proper functioning). The timer is calibrated for intervals of 0.1 and 0.5 second permitting use of intermediate time gradations. In the background are mouthgag headgear with disc electrodes and dish containing 25 per cent saline solution which is used to moisten the patient's temples to facilitate electrical conduction. Wires in the foreground lead to the electrodes when headgear is placed on patient.

1 The *infra-red ray* is the longest and varies from 770 to 15 000 millimicrons. The infra red ray is a source of heat. It does not penetrate the skin. Its therapeutic efficacy has been previously described (p 3787).

2 The *visible rays* range from the violet measuring 390 millimicrons to red at 770 millimicrons. These rays have no important therapeutic efficacy.

3 *Ultraviolet rays* extend from 136 to 390 millimicrons. They may be derived from solar energy or from various artificial sources. They produce photochemical effects such as activation of substances in the skin and blood stimulation of metabolism cellular activity growth and circulation.

BATHS

Methods of physical therapy by means of baths are summarized in Table 203

TABLE 204—PHYSICAL THERAPY BY ELECTRICITY

| Method | Technic | Remarks |
|--|---|---|
| Electric pad | See Local Application of Heat p 3791 | |
| Infra red ray | | |
| Diathermy | | |
| Hyperthermy | See p 3783 | |
| Faradism galvanism and sinusoidal currents | Specialist * | Test reaction of degeneration in muscles (p 2880) treatment of neuromuscular lesions |
| Electrolysis | Specialist † | Remove hair nevi and telangiectases |
| Iontophoresis | For ion transfer | With histamine and mecholyl (p 3873) produces vasodilatation of use in arthritis and peripheral vascular disease (p 1000) |
| Electrodesiccation | Specialist use of short wave diathermy for light tissue destruction monopolar current | Treats warts fibromas keratoses telangiectases and other local skin lesions |
| Electrocoagulation | Specialist use of high frequency extremely destructive bipolar current | Squamous cell epithelioma (p 3923) |
| Electrosurgery (cutting current surgical endothermy) | Similar to electrocoagulation but of lower voltage and milliamperage specialist use | Bladder and prostatic neoplasms (p 2322) |
| Electroconvulsions (electrofit) | Specialist ‡ | In psychoses (p 1323) |

The *interrupted galvanic current* is used to test the *reaction of degeneration* in muscles. Normally the cathodal closing contraction (CCC) is greater than the anodal closing contraction (ACC) the anodal closing contraction (ACC) is greater than the anodal opening contraction (AOC) the anodal opening contraction (AOC) is greater than the cathodal opening contraction (COC).

† For *electrolysis* and *epilation* a galvanic current of low voltage (20 volts) and low milliamperage (½ to 2 milliamperes) is used. The positive pole is connected to a sponge electrode which is held by the patient the negative pole has a fine platinum needle. For *epilation* the needle is inserted into the follicle alongside the hair shaft. Approximately 1 milliampere of current is allowed to flow for 15 to 30 seconds at the end of this time the hair root is destroyed and the hair may be removed with gentle traction. If the polarity is incorrect permanent black markings may be left in the skin.

‡ Electrically induced *convulsions* may replace convulsive therapy by insulin (p 1216) or metrazol (p 3570). Special technical equipment and experience are required the violence of the seizure may be mitigated by the use of intocostrin a derivative of curare (p 3588).

PHYSICAL THERAPY BY ELECTRICITY

The methods of physical therapy which utilize electricity are summarized in Table 204

Radiosensitivity—In the order of their radiosensitivity the susceptible cells are the lymphocytes polymorphonuclear and eosinophilic leukocytes basal epithelium of the salivary glands testes and ovaries basal epithelium of the skin mucous membranes stomach and small intestines alveolar epithelium of lungs and bile ducts and the epithelium of the tubules of the kidneys The more highly differentiated cells display greater resistance to radiation whereas tumors derived from embryonal tissue are extremely sensitive

Indications for Roentgen or Radium Therapy—The principal indications for roentgen or radium therapy are as follows

- 1 To hasten resolution of local infection—Pyoderms (p 3248) carbuncles (p 3249) parotitis (p 1708) tuberculous adenitis (p 3262) erysipelas (p 167)
- 2 To reduce endocrine activity—Of ovaries (Artificial Menopause p 2493) of testes (Male Characteric p 2414) of thyroid (p 1213) of thymus (p 1235) of anterior pituitary (p 1430)
- 3 To control overgrowth of lymphoid and myeloid elements—Leukemias (p 1100) Hodgkins Disease (p 1140) Status Thymolymphaticus (p 1235)



Fig 1118 --Rad odermatitis Left of face right, of breast

- 4 To control or destroy malignant growths—See Neoplasms (p 3199)
- 5 In dermatological therapy—*Congenital Lesions* (p 3145) Keratosis follicularis keratoderma palmaris et plantaris corns keloids lichen simplex *Neoplasms* (p 3199) Angiomas moles multiple benign cystic epithelioma leukoplakia keratoses precanceroses epitheliomas Paget's disease of breast melanocarcinoma sarcoma idiopathic multiple hemorrhagic sarcoma lymphosarcoma *Neurodermatoses* (p 3229) Localized neurodermatitis *Endocrinal Dermatoses* (p 3235) Senile vulvovaginitis *Metabolic Dermatoses* (p 3235) Lichen amyloidosis *Infections* (p 3245) Pyoderms especially carbuncles sycosis vulgaris axillary sweat gland abscesses tuberculosis verrucosa cutis scrofuloderma lupus vulgaris sarcoidosis warts dermatophytoses (superficial and deep) *Descriptive Dermatoses* (p 3355) Acne vulgaris granuloma annulare granuloma fungoides leukemia cutis lichen planus psoriasis parapsoriasis seborrhea hyperidrosis bromidrosis

In *tuberculosis* the use of ultraviolet irradiation requires considerable care and experience. In exudative tuberculosis toxic manifestations may be produced. These include headache, restlessness, insomnia, nervousness, irritability, elevation of the temperature and pulse rate with general activation of the symptomatology and signs. Employed with caution in *lymphatic tuberculosis*, *tuberculous peritonitis* and *enteritis tuberculosa*, *pleuritis* and *nonexudative manifestations of pulmonary tuberculosis*, in *tuberculosis of the bones and joints*, the *genito-urinary* and *pelvic organs* and the *larynx*, the results are usually most favorable.

In any instance ultraviolet irradiation is approached with great caution in the treatment of tuberculosis. By the Rollier method of progressive exposure to sunlight only the feet are exposed for 5 minutes of the first day. The legs are included in the exposure on the second day, the thighs on the third, the abdomen on the fourth and the chest on the fifth day. Using the artificial sources of ultraviolet irradiation the patient may be tested cautiously in a similar manner to adjudicate the individual response.

Contraindications—Ultraviolet irradiation is distinctly contraindicated in the progressive exudative forms of pulmonary tuberculosis, in tuberculosis of the adrenal gland, lupus erythematosus, pellagra, hyperthyroidism and diabetes.

Cold Quartz Light—Cold quartz light is a form of ultraviolet but of shorter wave length. It produces less intense erythema but desquamation is more marked. Its most important use is in conditions benefited by peeling of the skin. These include indurated *acne* (p. 3358), *pytiasis rosea* (p. 3410) and *tinea versicolor* (p. 3300).

ROENTGEN RAY AND RADIUM THERAPY

Roentgen ray and radium therapy are employed only by the competent specialist who may use the x-ray tube, radium emanations or radon.

Technics of Treatment—There are a variety of methods for use in treatment with radium or roentgen rays. In *massive dose technic* the total radiation is delivered at a single sitting but may be repeated after an interval of six weeks. In *saturation dose technic* the single massive dose is followed by small doses sufficient to compensate for tissue loss. The *divided dose method* (Coutard) provides for repeated small applications at short intervals.

Radium therapy may be employed for accessible areas. Under these circumstances the radium salts or the glass capillary tubes of radon are introduced *within a body cavity*; they may be inserted *interstitially* or placed *on the skin* over the site of a deeper local lesion.

The Effects of Radiation—Radiation effects are dependent upon tissue sensitivity. The more susceptible structures reveal the more marked histologic changes. In a tumor, for example, the cytoplasm and nucleus become enlarged. Later the nucleus becomes hyperchromatic with subsequent disintegration, the stroma undergoes fibrosis and finally the neoplastic tissue is replaced by heavy strands of an avascular connective tissue. The blood supply is decreased after an initial vasodilatation; an obliterative endarteritis develops with eventual thrombosis and cicatrization.

CHAPTER 176

GENERAL PRINCIPLES OF PHARMACOTHERAPY

| | |
|--|-------------------------|
| Official Drugs | Drug Excretion |
| Nonofficial Drugs | Drug Concentration |
| Proprietary | Drug Action |
| Drugs | Synergism |
| The Pharmacy | Antagonism |
| The Food Drug and Cosmetic Act of 1938 | Sensitivity |
| Federal Regulations | Tolerance |
| The Prescription | Idiosyncrasy |
| Drug Dosage | Habituation and Allergy |
| Drug Administration | Toxicology |
| Drug Absorption | Poisoning |
| Drug Distribution Storage and Intermedicary Metabolism | List of Common Poisons |
| | The Poison Kit |

In the practice of pharmacotherapy the physician is aided by the pharmacist pharmaceutical chemist pharmaceutical manufacturer botanist chemist physiologist bacteriologist immunologist pharmacologist toxicologist by Federal agencies and by the Committees and Councils of the American Medical Association. He receives guidance from current literature experiences of colleagues brochures of ethical manufacturers and publications and committee reports of official investigating agencies.

Despite the vast scope of pharmacotherapeutic enterprises it is the final responsibility of the practitioner to prescribe or administer each preparation and observe the particular effects on his individual patient. The successful therapist must be prepared to shift his attack while in motion with the skill and agility of a broken field runner in a football game. To accomplish his therapeutic goal he increases the dose of the drug in those who exhibit tolerance, he decreases the dose in the sensitive, he changes preparations when there are evidences of idiosyncrasy, and he must recognize toxicologic phenomena and evidences of poisoning from overdosage, for with the possible exception of penicillin there is no therapeutic agency for good that has not potentiality for significant untoward manifestations.

To acquire competence in clinical pharmacology the physician requires a vast store of concrete information and familiarity with the general principles of pharmacology. The latter are briefly summarized in this introductory chapter.

OFFICIAL DRUGS

Official drugs are listed and described in the United States Pharmacopoeia and the National Formulary.

The Pharmacopoeia—The United States Pharmacopoeia describes the approved therapeutic agents used in medical practice. It defines the sources physiochemical properties and tests for identity purity and potency, and it specifies the methods of storage and packaging and the average therapeutic dose. It is the legal standard for drugs according to

Radiation Sickness—The toxic effects of radiation may be local or systemic. The irradiated area may reveal deep pigmentation, atrophy, telangiectases, ulceration and malignant degeneration. The contiguous structures may undergo inflammatory changes and deformities due to scar formation as exemplified by the pulmonary fibrosis consequent to irradiation of the chest for mammary cancer.

Constitutional manifestations are most often related to the digestive tract or the bone marrow. The patient complains of *anorexia*, *nausea* and *vomiting* and the hemogram (p 3696) reveals the presence of *anemia*, *leukopenia* or a relative *lymphocytosis*.

Patient Management During Radiation Therapy—The area to be irradiated must be cleared of local infection before exposure. With lesions of the mouth, infected teeth are extracted; oral hygiene is of vital importance in the prevention of osteomyelitis of the jaw bones. During treatment, skin areas must not be exposed to local medication, adhesive plaster, ointments containing heavy metal or photosensitive drugs. Frequent blood counts are required. A significant or progressive fall in the red or white counts demands interruption of therapy.

Many roentgenologists favor the administration of large doses of *calcium* (p 604), *ascorbic acid* (p 629), *thiamine chloride* (p 622), *liver extract* (p 1048), *niacin* (p 622) and *iron* (p 1048) in the attempt to prevent and lessen radiation sickness. An occasional *transfusion* (p 3118) may permit the continuation of therapy without interruption. Intramuscular injections of 50 mg. pyridoxine before each treatment also may decrease the incidence and severity of radiation sickness.

an antacid powder or tablet he commonly takes a proprietary. The mass production of the compressed tablet to be used as a carminative and antacid selling at a cost of five or ten cents per package constitutes efficient and economical therapeutics.

The local pharmacist has neither the time nor the facilities to prepare an effervescent analgesic preparation that can compete in pharmaceutical manufacture or economy with the popular proprietaries that are readily available in drug stores.

NOSTRUMS

The practitioner should not display any tolerance for the nostrums with which unscrupulous manufacturers exploit the lay public. Many useless ingredients are included merely for effect. The price of the preparation is beyond all reason and the efficacy is often due to the alcoholic content rather than the allegedly specific drugs that are included. The conscientious practitioner will lend his individual weight to the campaign of the Federal Government and the Council on Pharmacy of the American Medical Association to curb and eventually eliminate the unreliable and unethical pharmaceutical products and their sponsors.

THE PHARMACY

The practitioner can best insure the quality of the drugs and the accuracy of the individual dose by specifying the product of a reliable pharmaceutical manufacturing house. The drug should be ordered in the dosage in which it is sold to the local pharmacist. This is particularly important with drugs prescribed in exceedingly small doses or those that are biologically assayed. It is virtually impossible for the pharmacist to standardize digitalis. With the type of balance ordinarily used in the average drug store the measurement of a fraction of a grain can only be roughly approximated.

As a matter of convenience and accuracy solid prescriptions are preferable to liquid preparations. Working people can and will carry a pill, a capsule or a tablet. A bottle containing a fluid prescription may be unwieldy.

THE FOOD, DRUG AND COSMETIC ACT OF 1938

The sale of drugs in the United States is regulated by the Federal Government through the provisions of the Food Drug and Cosmetic Act of 1938. The act rigidly controls the standards, purity and composition of drugs and drug mixtures and designates the U. S. Pharmacopeia and the National Formulary as its official organs. The provisions of the law are enforced by the Food and Drug Administration of the Department of Agriculture.

The act (1) provides for Government control of useful drugs, (2) prohibits traffic in drugs and devices dangerous to health, (3) regulates the labeling and advertising of therapeutic and cosmetic preparations, (4) orders labeling of habit forming narcotic and hypnotic drugs, (5) compels listing of the names of active ingredients with their quantities and proportions, and (6) stipulates the packaging of official drugs as prescribed by the U. S. P. and N. F.

the provisions of the Federal Pure Food and Drug Act (1938) It is revised every ten years by a special committee of physicians pharmacologists and pharmacists assisted by numerous subcommittees and advisory boards The drugs listed in the pharmacopeia which conform to the published standards of purity and quality are labeled USP

The Formulary—The National Formulary is an official compilation of the formulas of USP preparations It contains standards and tests for identity quality and purity of the drugs used in the formulas and for certain other materials commonly used in pharmacy The volume is revised periodically by a committee of pharmacists representing the American Pharmaceutical Association Drugs appear in the National Formulary because they are commonly used therapeutic merit is not a prerequisite for inclusion

The Epitome—The Epitome of the United States Pharmacopeia and National Formulary is published by the American Medical Association for the guidance of physicians In it are included the official names of the drugs and preparations listed in the USP and NF and a number of useful therapeutic facts The volume should be in the reference library of each physician

Useful Drugs—Useful Drugs is a brief presentation of essential clinical facts concerning the more important drugs It is issued by the Council of Pharmacy and Chemistry of the American Medical Association and is designed to serve as a basis for the teaching of materia medica and therapeutics In the preface to the first edition (1913) it was predicted that an intelligent and critical use of these selected drugs will prove their general sufficiency and show that many drugs now discussed in textbooks are superfluous and that many newly discovered or widely exploited proprietary preparations have no advantage over those contained in this book

NONOFFICIAL DRUGS

To guide the physician in the use of patented or proprietary drugs the American Medical Association has established a Council of Pharmacy and Chemistry which functions as a fact finding body The rules of the Council are designed to protect the medical profession and the public against secrecy fraud and objectionable advertising in connection with patented medicinal articles

New and Nonofficial Remedies—The Council's publication *New and Nonofficial Remedies* (N.N.R.) contains descriptions of the physical and chemical properties tests for purity and dosage available preparations actions and uses of accepted substances The inclusion of a remedy however is not necessarily an endorsement or recommendation by the Council Supplements to the N.N.R. and individual reports of the Council appear throughout the year and are published in the Journal of the American Medical Association Since many valuable additions to pharmacotherapy appear in N.N.R. before inclusion in the USP the alert practitioner cannot afford to be without this publication and its supplements

LIBERITARIANS

It is absurd to pretend that the legitimate proprietaries have no place in medical practice When the physician himself is in need of a dentifrice

| | |
|--|---|
| numerus (No) | number |
| pillula (pil) | a pill |
| post (p) | after |
| post cibum (p.c) | after meals |
| pro re nata (prn) | according to circumstances or occasionally |
| quantum satis sufficit or sufficient (q s) | a sufficient amount as much as is necessary |
| (q s ad) | a sufficient amount to make |
| quaque die (q d) | every day |
| quique hora (q h) (q 4 h) | every hour every four hours |
| quater in die (q i d) | four times a day |
| secundum artem (s.a) | according to the practice |
| semis (Ss or ss) | one half |
| sine (s) | without |
| si opus sit (s o s) | if necessary |
| signa (S) | sign or label |
| statim (stat) | immediately |
| ter in die (t i d) | three times a day |

Adoption of Metric System.—Prescription writing has been simplified by the action of the Council on Pharmacy of the American Medical Association which has decided against perpetuation of the apothecary system. The metric system is now official for the United States Pharmacopoeia and publications of the American Medical Association. This change will cause some difficulty for those physicians who have been taught the apothecary system for their benefit the conversion tables that have been officially adopted are appended.

METRIC-APOTHECARY EQUIVALENTS
APPROXIMATE EQUIVALENTS FOR GENERAL USE

WEIGHTS

| Apothecary or Troy | Metric | Apothecary or Troy | Metric | Apothecary or Troy | Metric |
|-----------------------|--------|-----------------------|--------|-----------------------|--------|
| 1 ounce = 30 | Gm | 2½ grains = 0.16 | Gm | grain = 11 | mg |
| 4 drams = 12 | Cm | 2 grains = 0.13 | Gm | ½ grain = 8 | mg |
| 2½ drams = 10 | Gm | 1½ grains = 0.1 | Cm | 1 grain = 65 | mg |
| 2 drams = 8 | Cm | 1 grain = 6 | mg | grain = 54 | mg |
| 75 grains = 5 | Gm | ½ grain = .30 | mg | grain = 4 | mg |
| 1 dram = 4 | Cm | ¼ grain = 4 | mg | grain = 32 | mg |
| 1 grain = 2 | Gm | ½ grain = 32 | mg | grain = 2 | mg |
| 30 grains = 2 | Gm | ¼ grain = 24 | mg | grain = 1 | mg |
| 15 grains = 1 | Gm | ½ grain = 2 | mg | ½ grain = 0.65 | mg |
| 10 grains = 0.65 | Cm | ¼ grain = 16 | mg | grain = 0.54 | mg |
| 7½ grains = 0.5 | Gm | | | ½ grain = 0.4 | mg |
| 7 grains = 0.45 | Gm | | | ¼ grain = 0.3 | mg |
| 6 grains = 0.4 | Gm | | | ½ grain = 0.26 | mg |
| 5 grains = 0.32 | Gm | | | ¼ grain = 0.2 | mg |
| 4 grains = 0.25 | Gm | | | ½ grain = 0.1 | mg |
| 3 grains = 0.2 | Gm | | | | |

FEDERAL REGULATIONS GOVERNING THE SALE OF SERUMS, VACCINES AND TOXINS

The importation, exportation and interstate sale of viruses, serums, vaccines and similar biological products are forbidden by Federal law unless the manufacturer holds a license issued on the recommendation of the United States Public Health Service. The law requires each package of biologic products to be marked with a date of expiration. Prior to the release of each lot, potency tests are made at the National Institute of Health for the following products:

Antitoxins

- 1 Botulinus
- 2 Diphtheria (also toxin and antitoxin)
- 3 *Cl. histolyticum*
- 4 *Cl. oedematiens*
- 5 Staphylococcus
- 6 Tetanus
- 7 Scarlet fever streptococcus
- 8 *Cl. welchii* (*B. perfringens*)
- 9 *Isbrio septicus*

Toxoids

- 1 Diphtheria
- 2 Tetanus

Antisera

- 1 Antidysenteric
- 2 Antimeningococcic
- 3 Antipneumococcic and type specific

Bacterial Vaccines

- 1 Paratyphoid A and B
- 2 Typhoid

THE PRESCRIPTION

The prescription is the physician's written order to be dispensed by the pharmacist. It includes the name, address and age of the patient, the drugs to be given with their respective amounts, the directions to the pharmacist, the directions to the patient and the signature of the prescriber.

Prescriptions should be clearly written in ink. It is good practice to file a copy of the prescription in the patient's chart since there are frequent demands for repetition of recipes that have proven particularly helpful.

The prescription should be written in English. The use of Latin is outmoded although certain abbreviations are retained for convenience. Among those most commonly employed are:

℞ (recipe)
ad libitum (ad lib)
ana (aa or aa)
ante (a)
ante cibum (a c)
aqua
aqua destillata
bis in die (b i d)
capsula (cap)
chartula (chart)
cum (c)
fac fiat fiant (f ft)

gutta guttae (gtt)
hora (h)
misce (M)
misce et divide in (M et D)
misce et fac solutionem (M et
 F sol)
misce et pone in (M et P)
ne repetatur (ne rep)

receive thou
 at pleasure
 of each
 before
 before meals
 water
 distilled water
 twice daily
 capsule
 powder
 with
 make let be made let them be
 made
 a drop drops
 hour
 mix
 mix and divide into
 mix and make a solution
 mix and place into
 do not repeat

Nonofficial or extemporaneous prescriptions whose popularity is definitely waning are composed of a *principal drug* or *basis* which gives the prescription its chief action a *subsidiary adjuvant drug* which augments the action of the principal a *subsidiary corrective drug* which offsets any undesirable action of the principal and a *vehicle* which is used as a solvent or to increase bulk.

After each drug listed in the body of the prescription the *total required amount* is noted in *metric system*. To avoid confusion in the decimals it is good practice to draw a line through the decimal point. The sum total of drugs prescribed is determined by the size of the individual dose and the number of doses that seems necessary. With solid prescriptions it is economical and useful to order the number of products contained in an original package as put out by the manufacturer. Ordinarily unit products are marketed in 40's 50's or 100's. For liquid preparations custom has sanctioned the use of bottles containing 30 cc 60 cc 120 cc 240 cc and 500 cc.

Directions and Signature—The directions to the pharmacist should be simple. Directions to the patient should be written clearly and fully without abbreviations so as to avoid any possible confusion and misunderstanding. It is necessary to state the *individual dose* the *frequency* with which it is to be repeated the *relationship to meals* and the *method of administration*. After checking the completed prescription the signature and narcotic registry number are added.

Incompatibility—Incompatibility may be chemical physical pharmaceutical or pharmacological. Substances may interact be insoluble immiscible or mutually antagonistic in action.

Some of the common incompatibilities are undernoted.

- 1 Acids and carbonates liberate carbon dioxide
- 2 The alkaline salts of organic acids precipitate salts of heavy metals
- 3 Alkaloidal salts are insoluble in strongly alcoholic solutions
- 4 Alkaloids and alkaloidal salts may be precipitated out of solution by hydroxides carbonates iodine bromides iodide salicylates benzoate borate metallic salts or tannic acid
- 5 Gums albumin and many inorganic salts are precipitated by alcohol from watery solutions
- 6 Hydroxides and carbonates in aqueous solutions precipitate alkaloidal salts
- 7 The addition of alkali to chloral hydrate may produce chloroform
- 8 Bromide chloride and iodide are apt to precipitate alkaloidal salts out of aqueous solutions
- 9 Chloral hydrate rubbed with camphor or menthol deliquesces
- 10 Glucosides are incompatible with alkalis mineral acids enzymes
- 11 Gums are precipitated by alcohol
- 12 Iron salts are incompatible with tannic and gallic acids carbolic acid salicylates hydroxide carbonates solutions of acacia
- 13 Mercury salts have so many incompatibilities that they should be prescribed alone. They precipitate alkaloid glucosides protein and salts of other metals
- 14 The salts of heavy metals are incompatible with alkaloids tannin protein acacia and a large number of organic substances

LIQUID MEASURES

| Apothecary | Metric | Apothecary | Metric | Apothecary | Metric |
|--------------------------|--------|------------------------|--------|---------------------|--------|
| 1 pint = 480 cc | | 4 fluid drams = 15 cc | | 20 minims = 12 cc | |
| 12 fluid ounces = 360 cc | | 2½ fluid drams = 10 cc | | 16 minims = 1 cc | |
| 8 fluid ounces = 240 cc | | 2 fluid drams = 7½ cc | | 15 minims = 0.9 cc | |
| 6½ fluid ounces = 200 cc | | 80 minims = 5 cc | | 12 minims = 0.75 cc | |
| 4 fluid ounces = 120 cc | | 65 minims = 4 cc | | 10 minims = 0.6 cc | |
| 3½ fluid ounces = 100 cc | | | | | |
| 2 fluid ounces = 60 cc | | 1 fluid dram = 3.7 cc | | 8 minims = 0.5 cc | |
| 1½ fluid ounces = 50 cc | | 50 minims = 3 cc | | 5 minims = 0.3 cc | |
| 1 fluid ounce = 30 cc | | 45 minims = 2.8 cc | | 3 minims = 0.19 cc | |
| ½ fluid ounce = 25 cc | | 32 minims = 2 cc | | 1½ minims = 0.1 cc | |
| 5½ fluid drams = 20 cc | | 30 minims = 1.8 cc | | 1 minim = 0.06 cc | |

FOR FAIRLY ACCURATE CONVERSION

| | |
|-----------------------------------|-------------------|
| 1 Gm = 15.43 | grains |
| 1 Gm = 0.2572 | dram |
| 1 Gm = 0.03215 | Troy ounce |
| 1 Gm = 0.03527 | Avoirdupois ounce |
| 1 Gm = 0.0022 | Avoirdupois pound |
| 1 grain = 0.0648 | Gm |
| 1 grain = 64.8 | mg |
| 1 dram = 3.858 | Gm |
| 1 Troy or Apothecary ounce = 31.1 | Gm |
| 1 Avoirdupois ounce = 28.35 | Gm |
| 1 Avoirdupois pound = 453.6 | Gm |
| 1 cubic centimeter = 16.23 | minims |
| 1 milliliter = 16.23 | minims |
| 1 milliliter = 0.2705 | fluid dram |
| 1 milliliter = 0.0338 | fluid ounce |
| 1 milliliter = 0.00211 | pint |
| 1 milliliter = 0.000264 | gallon |
| 1 minim = 0.06161 | cc |
| 1 fluid dram = 3.6966 | cc |
| 1 fluid ounce = 29.57 | cc |
| 1 pint = 473 | cc |

The Parts of a Prescription—The prescription proper consists of the superscription the body of the inscription the directions to the pharmacist or subscription the directions to the patient and the signature. There is an increasing and wholly laudable tendency to simplify prescription writing. As pharmacotherapy has become a more exact science there seems lesser need to complicate the prescription with Latin terminology combinations of multiple drugs many of which are quite completely inert and the archaic symbols of the apothecary system.

Superscription—The superscription includes the patient's name and address and age the date and the symbol *R* for recipe meaning take thou.

Inscription—The body of the prescription contains the name and the amount of each ingredient or the name and amount of an official recipe from the USP or NF.

Nonofficial or extemporaneous prescriptions whose popularity is definitely waning are composed of a *principal drug* or *basis* which gives the prescription its chief action a *subsidiary adjunct drug* which augments the action of the principal a *subsidiary corrective drug* which offsets any undesirable action of the principal and a *vehicle* which is used as a solvent or to increase bulk.

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- 3 Alkaloidal salts are insoluble in strongly alcoholic solutions
- 4 Alkaloids and alkaloidal salts may be precipitated out of solution by hydroxides carbonates iodine bromides iodide salicylates benzoate borate metallic salts or tannic acid
- 5 Gums albumin and many inorganic salts are precipitated by alcohol from watery solutions
- 6 Hydroxides and carbonates in aqueous solutions precipitate alkaloidal salts
- 7 The addition of alkali to chloral hydrate may produce chloroform
- 8 Bromide chloride and iodide are apt to precipitate alkaloidal salts out of aqueous solutions
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- 10 Glucosides are incompatible with alkalis mineral acids enzymes
- 11 Gums are precipitated by alcohol
- 12 Iron salts are incompatible with tannic and gallic acids carboic acid salicylates hydroxide carbonates solutions of acacia
- 13 Mercury salts have so many incompatibilities that they should be prescribed alone. They precipitate alkaloid glucosides protein and salts of other metals
- 14 The salts of heavy metals are incompatible with alkaloids tannin protein acacia and a large number of organic substances

- 15 Volatile oils in alcoholic solutions are thrown out of solution by dilution with water
- 16 Fixed oils form soap with hydroxide and metal oxide
- 17 Oxidizing substances such as chlorates permanganate peroxide chromic acid and nitrates may explode particularly if triturated in a dry state with an easily oxidizable substance such as glycerin hydrochloric or sulfuric acid
- 18 Resins are precipitated from alcoholic solution if sufficient water is added

Incompatibility is avoided by prescribing the important and potent drugs in the form in which they are distributed by the manufacturer

DRUG DOSAGE

The *correct dose* of a drug in the clinical sense is the amount that must be administered in order to obtain the desired therapeutic effect without the production of untoward manifestations. A *toxic dose* is one which gives rise to untoward effects. A *lethal dose* results in the death of the individual patient. The *therapeutic index* is calculated by comparing the therapeutic dose with toxic and lethal doses.

Average Dose—In administering a drug the physician is guided by the *average therapeutic dose* as given in the USP, NF or N.N.R. He must be prepared to increase the quantity if the desired beneficial effect is not obtained and toxic symptoms are not encountered; he must be ready to give a lesser dosage if his patient exhibits unusual sensitivity.

Individual Dose—The use of each drug for each individual patient is a new and fascinating experiment. If idiosyncrasy is feared the initial dose must be small and probatory as with *quinidine* (p. 861); if the patient exhibits unusual tolerance the dose must be increased; if the patient shows evidences of toxic manifestations before the therapeutic effect is seen the dose is decreased or the drug is discontinued. If a patient fails entirely to respond as in drug fastness an attempt is made to ascertain the cause of the failure by considering the factors of disease, absorption, metabolism, excretion and tolerance.

Dosage Tables—Attempts to simplify the problem of drug dosage by mathematical formulas are futile and may be misleading. Tables of dosage based on body weight in the adult have no particular value; the practitioner may as well inquire how much ether is required for surgical anesthesia, how many drinks it takes to get drunk, or how many analgesic tablets are needed to control a headache. There is no master key to successful pharmacotherapy; there is no substitute for a sound knowledge and keen observation.

DRUG ADMINISTRATION

Prior to the introduction of the hollow needle drugs were administered by topical application or the oral route. With the perfection of parenteral methods and the purification of drugs pharmacotherapy became a more exact and satisfactory science and a variety of avenues by which the drug might be introduced into the body became available for the ingenious practitioner.

DRUG ABSORPTION

When drugs are given by any route other than intravenously the factor of absorption is an important determinant of the intensity and duration of therapeutic activity. Thus in *sulfonamide therapy* (p 88) the rapidly absorbed compounds (sulfanilamide sulfadiazine) produce effectual blood concentrations in the treatment of systemic infection. The poorly absorbed preparations as exemplified by sulfaguanidine are utilized for their local activity on the contents of the intestine and are not intended for the accomplishment of high levels in the circulating fluids.

Absorption from the Skin and Mucous Membranes—Absorption from the skin usually requires a suitable fatty vehicle for penetration of the cuta-

TABLE 206—ROUTES FOR DRUG ADMINISTRATION

| Method | Indications |
|------------------------------|--|
| Topical application (p 3112) | Local effects occasional systemic effect after absorption of mercury androgen or estrogen |
| Iontophoresis (p 3192) | For ionized drugs using galvanic current |
| Inhalation | For gaseous and volatile drugs such as ammonia general anesthetics (p 324) nitrites (p 339) oxygen helium and carbon dioxide (p 336) and trichlorethylene (p 3264) |
| Nebulization | Of antibiotics in respiratory infection |
| Oral | Most frequently used for safety convenience and economy use solids or liquids give irritants after meals enteric coating for preparations digested by gastric juices and for irritants |
| Rectal | Suppositories or starch suspensions when stomach is intolerant use double oral dose |
| Sublingual | For nitroglycerin (p 339) androgen (p 2401) estrogen (p 2575) and progesterone (p 2517) |
| Intracutaneous (p 3770) | For allergens and vaccines |
| Subcutaneous (p 3370) | For water-soluble non-irritant drugs and biologicals for implantations of hormone pellets (p 3771) |
| Intramuscular (p 3772) | For more rapid effects of water-soluble drugs for irritant water-soluble drugs for suspensions of insoluble drugs and biologicals |
| Intravenous (p 3731) | For water-soluble drugs and biologicals only for rapid assured absorption high concentration and minimum local irritation Beware of local and systemic reactions (p 3771) |
| Intrathecal (p 3782) | For local effects on meninges and cord only water-soluble drugs and biologicals |
| Intracardiac (p 3779) | For emergency only |

neous glands. Rapidity of absorption is favored by rubbing the agency into a wide vascular area.

Mucous membrane absorption is practiced in the *sublingual region*. A soluble preparation such as *nitroglycerin* may be absorbed with a rapidity that approaches the accomplishment of intravenous injection. Sublingual medication is also utilized for hormonal effects with *androgen*, *estrogen* and *progesterone*.

Absorption from the Digestive Tract—Most drugs that are given orally are absorbed from the small intestine. Often the stomach is a barrier to pharmacologic activity; in the presence of vomiting the drug is ejected; in gastric dilatation the preparation is held within the distended viscus; biological products such as organ extracts and penicillin undergo digestion and inactivation by the gastric juices.

Rapidity of absorption is enhanced by the free water solubility of the preparation and the fasting state in the gastro intestinal tract. For delayed absorption products are given with enteric coating and are prescribed after meals.

Failure of drug action may be due to faulty absorption from the digestive tract. At times the imperfection is a highly individual phenomenon that cannot be predicted or explained. More often it is due to a tangible disturbance such as vomiting, retention in a dilated stomach, edema of the intestinal mucous membrane (congestive heart failure), absence of bile from the intestinal tract (with impaired absorption of fat soluble vitamins A, D and K), rapid evacuation of the drug (diarrheal conditions) and impermeability of the wall of the bowel in lesions such as sprue and celiac disease.

Absorption from the Subcutaneous and Muscular Tissues—The rapidity of absorption from the subcutaneous and muscle tissues depends upon the physicochemical properties of the drug and the local blood supply of the injected area. Water soluble drugs are absorbed rapidly, the speed varying directly with vascularity, fat soluble drugs are absorbed more slowly and act over a longer period of time. The injection of insoluble suspensions that dissolve slowly in the body fluids favors prolonged action, as exemplified by *protamine zinc insulin* in diabetes mellitus (p. 1246) and *pitresin tartrate in oil* in diabetes insipidus (p. 1180).

Because of better vascularity there is more rapid absorption from muscles than from the subcutaneous tissues. Absorption may be hastened by the application of heat or by massage; it is delayed by the simultaneous administration of *epinephrine* which causes local vasoconstriction, as in local anesthesia, and impedes the removal of the anesthetic from the injection site.

THE DISTRIBUTION, STORAGE AND INTERMEDIARY METABOLISM OF DRUGS

After absorption, drugs are distributed throughout the body by way of the circulating fluid. Preparations which readily penetrate cell membranes are evenly divided in the extracellular and intracellular fluids. Agencies which display an *elective affinity* appear in higher concentrations in specific tissues; other substances seem excluded from cell penetration by an effectual barrier. The pharmacology of the sulfonamides best illustrates these principles. *sulfanilamide* appears to be evenly distributed throughout the body and is present in almost equal concentrations in blood and spinal fluid. *sulfathiazole* does not penetrate the pia arachnoid barrier and is demonstrable in the spinal fluid in low concentrations. *sulfaguanidine*, present in high concentrations within the lumen of the bowel, is represented by traces in blood and urine.

Drug distribution is influenced by *pathological conditions*. In congestive heart failure, large amounts of *digitalis* (p. 855) may accumulate in edema fluid, when the latter is mobilized by diuresis, the tissues may become flooded with the drug and evidences of poisoning are produced.

Specific Affinity—The specific affinity of particular tissues for certain drugs may be related to therapeutic activity. The *volatile anesthetics* (p. 3924) gain quick access to the lipid rich cells of the central nervous

system and produce depression. According to the Meyer Overton law there is direct parallelism between the affinity of the general anesthetic for lipids and its depressant action.

Storage—The factor of storage has pharmacologic significance. Depletion of the store of vitamin K in the liver causes hemorrhagic disease of the newborn (p 1113). The precipitation of stored lead from bone when there is an increased demand for calcium may produce the manifestations of *plumbism* (p 762).

Intermediate Metabolism—Drug action may be dependent upon intermediate metabolism within the body tissues. *Arsphenamine* (p 116) is not spirocheticidal in the test tube but becomes a potent therapeutic agent upon conversion within the body to arsenoxide. *Quinine* (p 516) is plasmodicidal only when it has become activated after contact with living cells.

Changes in intermediate metabolism may have noxious effects. Patients with chronic liver disease may develop a deficiency syndrome due to in-



Fig 1119—Bromide eruption in nursing baby whose mother was taking bromides

ability of the damaged cell to convert carotene into vitamin A. The patient with liver damage is hypersensitive to *barbiturates* and *opiates* because of their imperfect destruction.

EXCRETION OF DRUGS

Most drugs are metabolized in some unique manner by contact with the tissues. Others are excreted unchanged by way of the urine, feces, expired air or sweat. Rapidly excreted drug (*penicillin*) require frequent dosage in order to maintain an effective blood concentration. Slowly excreted drugs (*digitalis*) may cause cumulative poisoning unless the dosage is reduced after the desired therapeutic effect has been attained.

If the principal excretory channel is obstructed the therapeutic usefulness of the compound may be impaired while the tendency to toxicity is increased. Patients with renal insufficiency react badly to drugs that are

normally excreted in the urine. With cessation of respiration during the administration of a volatile anesthetic the maintenance of artificial breathing is required for tissue nutrition and also to hasten the elimination of the drug.

The concentration of drug in the organ of excretion may lead to local injury. The kidneys often suffer serious damage as the result of the urinary excretion of *sulfonamides* which tend to crystallize and form calculi (*urethralias medicamentosa*). The renal excretion of *mercury* may cause *anuria*; its excretion in the feces may produce an *ulcerative colitis* and its appearance in the saliva may give rise to a *necrotizing gingivitis*.

A knowledge of the excretory pathway of a particular drug is often of value in *diagnosis*. The presence of alcohol, barbiturate, lead, arsenic and *mercury* in the urine may suggest a poisoning. The excretion of organic iodide derivatives is the basis for intravenous urography (p. 2251) and oral cholecystography (p. 2000).

Placental Transfer Lactation—Many drugs gain access to the fetal circulation by their ability to cross the placental barrier. Depressants of the central nervous system such as *morphine* and *barbiturates* must be used carefully during childbirth. One of the great disadvantages of painless labor is the high incidence of *asphyxia neonatorum* (p. 2766). On the other hand the ability of organic *arsenicals* to enter the fetal circulation is responsible for the effective prevention of *congenital syphilis* in babies born of leucotic mothers (p. 2787).

During lactation many drugs are excreted in the milk. Sufficient *brocade* (Fig. 1119) and *iodide* may be administered to a nursing to cause a *dermatitis medicamentosa*.

DRUG CONCENTRATION

The concentration of a drug within the tissues depends upon the sum of the factors of *absorption*, *distribution*, *detoxification*, *inactivation* and *excretion*. Much of the success of *sulfonamide* medication is dependent upon studies of effective levels. High blood level is required for systemic infections; the optimum for urinary antiseptics is a relatively low blood level and a high concentration in the urine. The goals in intestinal infections are low blood and urine levels with high fecal concentrations (p. 100).

In general the factors that produce low or ineffectual concentrations are imperfect absorption, poor distribution and rapidity of detoxification, inactivation and excretion. For example, *epinephrine* is instantly absorbed and rapidly distributed so that an effectual concentration is soon achieved. Unfortunately the drug is rapidly oxidized so that the pharmacologic effect is completed within a few seconds to a minute and repetition of the dose may be required within a short time. In contrast to this, arsenic accumulates slowly even when given intravenously. A high and effectual blood concentration may not be accomplished for several days even during the course of the continuous intravenous drip. A high level must be sustained if effective spirocheticidal activity is to be achieved but since excretion is also slow, cumulative poisoning may penalize the enthusiastic therapist and his patient.

The factors which influence concentration are themselves variables.

adding to the difficulty of conducting the therapeutic experiment Absorption is impaired when there are local circulatory difficulties so that pharmacologic activity is feeble when a subcutaneous injection is given to a patient whose tissues are edematous In contrast high concentrations may be produced inadvertently with an undesirable increase in toxicologic phenomena when *heavy metals* *barbiturates* or *morphine* are administered to the patient whose kidney or liver functions have become insufficient Since it is difficult to obtain definitive data relative to drug concentration the practitioner is compelled to rely on *clinical observation* and *experience* in his efforts to obtain maximum benefits for his patient with minimum untoward reactions

DRUG ACTION

The action of a drug is the modification of function that it induces Most actions consist of *stimulation* or *depression* of some existent functions No known drug causes a particular organ to assume a new function

The action of a drug must be distinguished from its effects Either is a depressant of the central nervous system and the effect of this action is *general anesthesia* Epinephrine stimulates motor nerve endings of the smooth muscle of the arterioles and the effect is *vasoconstriction* with a rise in blood pressure If the action of a drug and the function of the involved tissue are known the effect can be deduced

Pharmacodynamic Variables —Drug action is modified by many variables one of the most important of which is the *functional capacity* of the tissue upon which the preparation acts The cardiac response to digitalis (p 855) may be beneficial or noxious dependent upon the tonus of the myocardium A *mercurial diuretic* is of little value in the presence of renal insufficiency and a medulla depressed to the point of respiratory paralysis by an overdosage of barbiturates cannot be expected to respond to the action of medullary stimulants

Some drugs are useful only in the presence of disordered function *Antipyretics* (p 2832) lower body temperature only in the febrile many *vitamins* and some *hormones* have valuable therapeutic actions only in the presence of a specific deficiency syndrome The presence of a *pathological lesion* may impair drug action The patient with the nephrotic syndrome is resistant to thyroid extract and little can be accomplished by digitalis when there is extensive myocardial scarring or myomalacia

Drug action may be influenced by the factor of *dosage* Small quantities of epinephrine produce vasodilatation whereas larger amounts reveal the more powerful vasoconstrictor effect Therapeutic doses of a *mercurial* are actively diuretic while toxic amounts produce anuria

Synergism —The simultaneous administration of drugs which have the same or a related action may result in a *summation* or a *potentiation* Illustrative of the latter is the pressor response to the combination of cocaine and epinephrine when given together these drugs elevate blood pressure much more than the sum of the two drugs given separately

Antagonism —Drugs given in combination may limit or neutralize one another Antagonism or *negative summation* is the opposite of synergism Depressants and stimulants of the central nervous system are obvious antagonists Very often an antagonist is given to protect against some un-

desirable action of the principal drug. The use of *atropine* with *morphine* affords some measure of protection against the respiratory depression produced by the latter. The *barbiturates* are used to prevent *cocaine* reactions and *ephedrine* combats the undesirable hypotension that occurs during spinal anesthesia.

Sensitivity—An increase in the quantitative response to the administration of a drug is encountered in sensitivity. Drug sensitivity is distinct from idiosyncrasy (p. 3813) in that the reaction is characteristically pharmacodynamic and is not altered qualitatively.

Drug sensitivity may cause major tragedies in clinical practice. Infants and elderly individuals may develop marked respiratory depression from therapeutic doses of an *opiate*. An occasional patient dies instantly when injected with *cocaine* or a *cocaine derivative*. A small dose of *epinephrine* may cause violent sympathomimetic manifestations in *hyperthyroidism* and *acute belladonna poisoning* may result from a single drop of a mydriatic instilled into the conjunctival sac.

When drug sensitivity is encountered the practitioner is often held culpable, the suspicion of overdosage being difficult to combat. Those physicians who have themselves had experience with drug sensitivity are most likely to be tolerant when judgment is passed on a more recent victim. He jests at scars that never felt a wound.

Tolerance—An interesting and often disconcerting aspect of drug action is the failure of an occasional patient to react to a drug given in the usual dosage. *Insensitivity* or *refractoriness* may be acquired by the habitual use of cathartics or alcohol. If it is innate, it is noted the first time the remedy is taken. Many normal individuals are extremely tolerant to large doses of *thyroid extract*; some patients require incredible amounts of inhalant anesthetic before they are well relaxed during a surgical procedure.

The phenomenon of tolerance is encountered after the administration of *hormones* in diseases of the endocrine glands. Some diabetics become temporarily resistant to *insulin* and require extreme amounts before the disease is controlled (p. 1246). *Refractoriness* to *pituitary antidiuretic hormone* is occasionally encountered in *diabetes insipidus* (p. 1180), and a diminishing response to repeated administration (*tachyphylaxis*) limits the therapeutic value of *parathyroid* (p. 726) and *anterior pituitary extracts* (p. 1169). These effects may be due to the production of *neutralizing antibodies* or *antihormones*.

Fastness—Tolerance is an important limiting factor in the success of chemotherapeutic agents. The invading parasite may become refractory to the chemical agent and is then capable of existing in the presence of adequate concentrations of the drug in the body. Drug fastness or resistance is encountered with *sulfonamide* in *gonorrhea* (p. 217), *quinine* and *atabrine* in *malaria* (p. 507) and *arsenic* in *sypylis* (p. 331). The resistance of pathogenic organisms to efficient chemotherapeutic agents constitutes a major problem of present day pharmacology.

Immunity—From an immunologic standpoint, acquired tolerance is a therapeutic aim. The ability of the body to resist the injurious actions of bacteria is built up gradually by the injection of vaccines, toxin antitoxin mixtures and toxoids. It is not improbable that a related immune mechanism is the basis for tolerance to most drugs. On this account, it is

our practice to avoid the use of the potent sulfonamides for example unless the stakes are worth the hazard of the production of fastness

IDIOSYNCRASY

Drug idiosyncrasy is the abnormal or unusual response of any given individual to a drug. It is one of the most annoying and embarrassing situations of clinical practice. It is of such frequent occurrence that the busy practitioner encounters drug idiosyncrasy as an everyday experience. Drug idiosyncrasy seems unrelated to true allergy. Though it may be transitory as a general rule it is persistent.

There is no way in which drug idiosyncrasy may be anticipated or avoided. It has a habit of appearing at the most inopportune time and under the most unfortunate circumstances. So common is drug idiosyncrasy that the practitioner should always suspect its presence when fresh symptoms arise in those patients who have been recently given any new pharmacological preparation. Drug idiosyncrasy may arise from topical or systemic introduction of the therapeutic substance.

Contact Dermatitis—Illustrative of the idiosyncrasy due to topical application is the dermatitis that arises for example in dentists who handle various types of local anesthetics. Contact dermatitis is well known as the result of the use of cosmetics, exposure to chemicals in industry or from the therapeutic application of silver ointments, lotions or washes (p. 3330).

Dermatitis Medicamentosa—Dermatitis medicamentosa is a skin eruption produced by drugs taken internally and is one of the most commonly encountered skin conditions in private practice. The most frequently observed agencies include iodide, bromide, the coal tars, the arsenicals, the barbiturates and the sulfonamides (p. 3335).

Systemic Idiosyncrasy—Systemic idiosyncrasy is exceedingly frequent. Thus morphine may produce its characteristic narcotic effect in most individuals. On the other hand certain patients become excited exhibiting the so called cat action (p. 3858) characterized by restlessness, excitement and even mania.

Even the simplest drugs such as the commonly employed coal tars (*acetylsalicylic acid*) produce intractable vomiting in certain sensitive individuals. Aminopyrine causes fatal agranulocytosis under conditions that are not completely understood. Cinchophen may produce a fatal hepatitis though thousands of patients take the drug over long periods of time and in large doses with complete impunity. In any large series of patients receiving arsenotherapy for syphilis there will be encountered a certain number of untoward reactions. These may be manifest in the central nervous system (toxic encephalopathy), the bone marrow (aplastic anemia), the skin (dermatitis exfoliativa) or the liver (toxic hepatitis).

So far as is known today, idiosyncrasy cannot be predated, prevented or alleviated by any therapeutic procedure. In view of its frequency the careful practitioner will always note the effect of a small and probatory dose of important drugs before employing full therapeutic amounts. Even this precaution may not be sufficient since certain types of idiosyncrasy such as dermatitis exfoliativa or hemorrhagic encephalitis do not appear until considerable time has elapsed following the introduction of the drug.

Drug idiosyncrasy is a genuine and common hazard of pharmacology and therapeutics. Many of the greatest injustices heaped upon the innocent practitioner result from his encounters with untoward drug effects and idiosyncrasy. Patients rarely resent being told frankly of the possibility of



Fig 1190—*A* Atopic neurodermatitis (eczema) in adult female. Probably psychogenic. *B* Atopic dermatitis (eczema) in child. Of digestive etiology. *C* Contactual atopy from cosmetic (lipstick). *D* Contactual atopy from wearing apparel (hatband). *E* Contactual atopy from occupational contact with cosmetic (hair dye) in beauty shop operator. *F* Contactual dermatitis from drug (sulfathiazole ointment).

this situation. They may be forewarned provided that the suggestibility of the individual is not excessive. Patients who show drug idiosyncrasy or develop drug poisoning should be acquainted with this fact so that they may call it to the attention of other physicians.

HABITUATION AND ADDICTION

The continued use of certain drugs produces a psychic craving or dependence. *Habituatio*n may occur with any drug taken for pleasurable effect or the relief of tension, pain or insomnia. As a rule the dependence

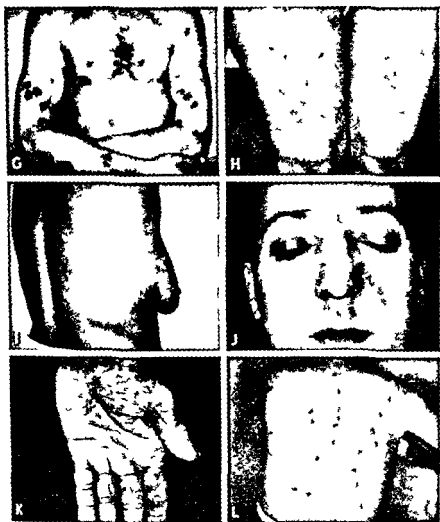


FIG. 1121.—G Drug atopy Dermatitis medicamentosa from phenolphthalein taken as cathartic. H Drug atopy Dermatitis medicamentosa from phenobarbital taken as sedative. I Drug atopy Dermatitis medicamentosa from arsenic injections for syphilis. J Contactual atopy (occupational) From refrigerating gas. K Contactual atopy (occupational) From tar. L Trichophytid from ringworm of feet.

is largely psychological; the patient is convinced of the indispensable value of the medication and withdrawal induces intense craving.

Addiction is an extreme degree of habituation and is characterized by psychic craving and organic withdrawal symptoms. The continued use of

the drug seems to produce a fundamental alteration in cellular processes and becomes a factor essential to normal performance. Addiction is encountered with *alcohol cocaine morphine morphine substitutes opium cannabis* (marihuana hashish) and *barbiturates*. The clinical manifestations of drug addiction and the various *abstinence syndromes* are elaborated in the discussion of the individual drugs.

Federal Regulations Pertaining to the Control of Narcotics—The Federal Narcotics Regulations require the physician who prescribes narcotics to obtain from the Collector of Internal Revenue a special tax stamp registry number and blanks. The registry number must appear on every prescription for a narcotic written by the physician. The narcotic order written in ink must give the name address and age of the patient as well as the reason for which the drug is given. If the drug is being prescribed in large doses to a patient with inoperable carcinoma the phrase *Article 83 Exception 1* is appended. If the patient is an addict whose life would be endangered by withdrawal the designation is *Article 83 Exception 2*. Narcotic prescriptions cannot be refilled by the pharmacist. Each year the physician is required to submit a complete inventory of narcotics on hand.

TOXICOLOGY

Toxicologic phenomena as opposed to poisonings (p. 743) arise during the course of the administration of the drugs intended for therapeutic purpose. The untoward symptoms may be side effects, the results of cumulation or overdosage through error.

Side effects—The side effects of a potent drug may be so severe as to lead to cessation of the intended therapeutic measure. Opium derivatives prescribed for the relief of pain may cause dangerous respiratory depression; a mercurial intended for anti-syphilitic therapy may be responsible for renal irritation and anuria.

Cumulation—Cumulation is seen with the slowly excreted drugs such as the metals or digitalis. The latter while exerting its benefits on the circulation may cause nausea and vomiting; the heavy metals may injure their excretory channels producing gingivitis colitis or a nephritis.

Overdosage—Overdosage is occasionally the result of an unfortunate error. A misplaced decimal point may cause death or serious damage; injection of a 10 per cent cocaine solution when 1 per cent was intended may prove disastrous; and the substitution of boric acid crystals for milk sugar in bottle formulas has caused the death of several infants in a hospital ward.

POISONING

Whereas toxicologic phenomena arise from the intended therapeutic administration of drugs, poisoning constitutes a *medicolegal problem* in instances where drugs have been taken as the result of *accident*, in attempted *suicide* or given with *criminal intent*. Additionally, *industrial poisoning* is a formidable problem with many ramifications in the domain of public health.

An entire section has been devoted to the problems of poisoning (p. 743) with emphasis on symptoms, diagnosis, methods of prevention and treatment. For present purposes tables are appended in which are

listed the common names of poisons (p 743) and the contents of an emergency poison kit together with indications for their use (p 3818)

TABLE 207—COMMON NAMES OF POISONS

| | |
|------------------------------------|--|
| Ant Paste | Arsenic trioxide calcium arsenate (p 752) |
| Ant Powd r | Sodium fluoride (p 755) |
| B ite Almond | Yields hydrocyanic acid (p 748) |
| Blue V t ol | Blue Stone or Blue Copper as Copper sulfate |
| Bordeaux Mixtu e | Copper hydroxide |
| Casto Beans | Ricin |
| Colon al Spi t or Columb an Spirit | Methyl alcohol (p 756) |
| Cre m of Tartar | Potassium acid tartrate |
| Fish ba r as | Picrotoxin (p 3870) |
| Har Dyes | Paraphenylened amine pyrogallol silver ammonium nitrate |
| Indian Tobacco | Lobelia (nicotine like) |
| Insectic des and Fast Po sons | Carbon disulfide chloropicrin copper arsenite or hydroxide cubeb or derris powder helichore hyd cyanic acid lead arsenate mercuric chloride methyl bromide naphthalene nicotine sulfate orthodichlorobenzene paradichloro benzene phenol pheoethazine pyrethrum quassia rotenone sodium arsenite or arsenate sodium fluoride thallium sulfate zinc phosphide |
| J mson or Jamestown Weed | Thorn or devil's apple stramonium (p 3875) |
| Knockout Drops | Chloral (p 3837) and alcohol |
| Lily of the Valley | Convallaria (digital s-like) (p 85) |
| Lye | Sodium hydroxide |
| Milk S ckness | Milk from cow poisoned with rayless goldenrod or richweed |
| M th Poison | Pyrethrum naphthalene |
| Mush rooms | Muscamine and other toxins (p 740) |
| Mussel Po on | Toxin of plankton dinoflagellate Gonyulax catenella |
| Obesity Cu s | Thyroid dinitrophenol dinitrocresol (p 698) |
| Oleand | Digitaloid glucosides (p 855) |
| Po son Oak or Ivy | Ilhus toxin toxicodendol (p 2330) |
| Pars Green | Copper acetoarsenite (p 752) |
| Rat or Rodent Po son | Barium carbonate red squill strychnine thallium sulfate (p 760) |
| Roach Po n | Sodium fluoride (p 755) |
| R d P c ptat | Red oxide of mercury (p 76) |
| Salt peter | Potassium nitrate (p 601) |
| S ltpete | Chile Sodium nitrate (p 3892) |
| Salts of Lemon or So rel | Potassium acid oxalate |
| Sche lers o Em ald Green | Copper arsenite (p 752) |
| Sheep Dip | Solution of saponated cresol (lysol) (p 757) |
| Span h Fly | Cantharides |
| Spray Residue | Lead arsenate (most common) phenothiazine and other insecticides |
| Tart | Emetic Antimony and potassium tartrate (p 732) |
| T mto Po son | Para or orthodichlo benzene |
| V triol or Oil of Vi ol | Sulfuric acid concentrated (p 759) |
| White A nic | Arsenous Trioxide (p 752) |
| White P c ptate | Ammoniated mercury (p 765) |
| Wood Al ohol | Methyl alcohol (p 756) |

From Cutting W C Manual of Clinical Therapeutics Philadelphia W B Saunders Company 1943

The common names of poisons often do not indicate the nature of the chemical ingredients for purposes of clarification Table 207 has been prepared for the information of the practitioner summoned to the patient who has been made ill as the result of chance or purposeful poisoning

TABLE 203—CONTENTS OF POISON ANTIDOTE KIT AND INDICATIONS FOR USE

| Preparation | Indication |
|--|--|
| Apomorphine hydrochloride 8 mg ($\frac{1}{4}$ gr) | Emetic of choice unless patient is unconscious |
| Stomach tube | For gastric lavage. Avoid if local irritant has been swallowed |
| Milk tea vinegar egg white and olive oil | Easily obtained for demulcent and protective actions. For protein and alkaloid precipitation |
| Universal antidote R Charcoal 50.0 Magnesium oxide 25.0 Tannic acid 25.0 Make powder | Combination adsorbent demulcent and precipitant |
| Potassium permanganate 1 per cent solution (30 cc) | Dilute with 20 parts of water for gastric lavage |
| Bicarbonate of soda ($\frac{1}{4}$ lb) | For gastric lavage if acid has been taken |
| Vinegar | For gastric lavage if alkali (lye) has been taken |
| Sodium formaldehyde sulfoxylate in ampoule (10 gm) | Make up to 500 cc with water and use as lavage in mercury poisoning. May give 200 cc intravenously if patient is seen within 1 hour of poisoning |
| Sodium amytal 0.3 gm (5 grains) in ampoules or pentothal sodium 1 gm (15 grains) in 20 cc of water | Give amytal intramuscularly or 4-6 cc of pentothal intravenously (p. 38-3) for excitant and convulsant poisons |
| Chloroform (glass-stoppered brown bottle and mask) | For inhalation in case of excitant and convulsant poisons |
| Morphine sulfate (15 mg $\frac{1}{4}$ grain) | Use cautiously with excitants and convulsants |
| Atropine sulfate 1 mg ($\frac{1}{16}$ grain) | Use cautiously in respiratory depression specific in mechoyl poisoning (p. 3573) |
| Caffeine-sodium benzoate 0.5 gm ($7\frac{1}{2}$ gr) | Questionable value as stimulant use cautiously if at all |
| Dimercaptol (BAL) | Specific value in arsenic and mercury poisonings (p. 767) |
| Nikethamide (coramin) and Metrazol 1-5 cc of 25 per cent solution | Questionable value as respiratory and circulatory stimulants use cautiously if at all |
| Picrotoxin ampoules (20 cc) 1 cc = 3 mg | Use cautiously intravenously in barbiturate poisoning avoid convulsions |
| Epinephrine hydrochloride 1:1000 | Use cautiously as emergency circulatory stimulant may be given by intracardiac injection (p. 3779) for resuscitation |
| Amyl nitrite in ampoule | Cyanide poisoning (p. 748) |
| Sodium thiosulfate 1 gm (15 grains) in ampoules | Intravenous injection in recent iodine arsenical and cyanide poisoning |
| Methylene blue (1 per cent solution in 18 per cent sodium sulfate) | Inject 50 cc intravenously for coal tar and cyanide poisonings |
| Penicillin | In granulopentias |

SUGGESTED ROUTINE IN MANAGEMENT OF POISONING

- 1 Send for oxygen and carbon dioxide tanks in *respiratory difficulties*
- 2 Send for pulmotor (police fire or gas departments) in *respiratory failure*
- 3 Remember *medicolegal aspects* Send for police on slight suspicion
- 4 Do not overtreat with *pharmacologic antidotes* such as caffeine strychnine picrotoxin morphine digitalis atropine coramine and epinephrine Do not substitute one type of poisoning for another
- 5 Inject BAL in arsenic and mercury poisonings (p 767)
- 6 Remember that *would be suicides* may try again Psychotherapy or institutionalization may be required (p 1327) in period of recovery
- 7 Seal and label *lavage returns urine and stool* for diagnostic or medicolegal examinations Locate possible *poison source* (bottles boxes etc) and retain as evidence
- 8 Do not sign *death certificate* if there is medicolegal aspect until authorities have investigated
- 9 In *corrosive poisoning* instrumentation (stomach tube) may cause perforation
- 10 *Comatose patients* may develop aspiration pneumonitis from emesis with apomorphine
- 11 Treat *shock* if present with sodium lactate orally (p 937) or intravenous plasma (p 3775)
- 12 Initiate prophylactic chemotherapy with penicillin (p 106) especially in respiratory and granulopenic conditions

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| Universal antidote R _x Charcoal 50.0 Magnesium oxide 25.0 Tannic acid 25.0 Make powder | Combination adsorbent, demulcent and precipitant |
| Potassium permanganate 1 per cent solution (30 cc) | Dilute with 20 parts of water for gastric lavage |
| Bicarbonate of soda ($\frac{1}{2}$ lb) | For gastric lavage if acid has been taken |
| Vinegar | For gastric lavage if alkali (lye) has been taken |
| Sodium formaldehyde sulfoxylate in ampoule (10 gm) | Make up to 500 cc with water and use as lavage in mercury poisoning. May give 200 cc intravenously if patient is seen within 1 hour of poisoning |
| Sodium amytal 0.3 gm (5 grains) in ampoules or pentothal sodium 1 gm (15 grains) in 20 cc of water | Give amytal intramuscularly or 4-6 cc of pentothal intravenously (p. 3813) for excitant and convulsant poisons |
| Chloroform (glass stoppered brown bottle and mask) | For inhalation in case of excitant and convulsant poisons |
| Morphine sulfate (15 mg $\frac{1}{4}$ grain) | Use cautiously with excitants and convulsants |
| Atropine sulfate 1 mg ($\frac{1}{64}$ grain) | Use cautiously in respiratory depression, specific in mecholyl poisoning (p. 3873) |
| Caffeine-sodium benzoate 0.5 gm ($\frac{7}{8}$ gr) | Questionable value as stimulant, use cautiously if at all |
| Dimercaptol (BAL) | Specific value in arsenic and mercury poisonings (p. 767) |
| Nikethamide (coramin) and Metrazol 1-5 cc of 25 per cent solution | Questionable value as respiratory and circulatory stimulants, use cautiously if at all |
| Picrotoxin ampoules (20 cc) 1 cc = 3 mg | Use cautiously intravenously in barbiturate poisoning, avoid convulsions |
| Epinephrine hydrochloride 1:1000 | Use cautiously as emergency circulatory stimulant, may be given by intracardiac injection (p. 3779) for resuscitation |
| Amyl nitrite in ampoule | Cyanide poisoning (p. 748) |
| Sodium thiosulfate 1 gm (15 grains) in ampoules | Intravenous injection in recent iodine, arsenical and cyanide poisoning |
| Methylene blue (1 per cent solution in 18 per cent sodium sulfate) | Inject 50 cc intravenously for coal tar and cyanide poisonings |
| Penicillin | In granulopenias |

Syrups—Syrups are solutions of 85 per cent sucrose in water plus a flavoring substance. They are excellent for masking the unpleasant tastes of water soluble drugs.

Mucilages—Mucilages are aqueous solutions of gums or the mucilaginous principles of vegetable substances. They are especially useful in the preparation of suspensions and emulsions. They mask unpleasant tastes by surrounding the particles of the dispersed phase of an emulsion or suspension by a fine film of the colloidal mucilage.

Elixirs—Elixirs are solutions containing 25 per cent alcohol. They may be used as solvents for specific drugs (medicated) or purely as diluents and vehicles (non medicated).

Tinctures—Tinctures are hydro alcoholic or alcoholic solutions of crude drugs prepared in a quantitative manner. As a rule tinctures of potent drugs contain 10 gm per 100 cc whereas those of less potent substances contain 20 gm per 100 cc.

Spirits—Spirits are alcoholic solutions of volatile drugs usually oils. They contain 10 per cent of the volatile flavoring agent. Spirits may be medicated (aromatic spirit of ammonia) or non medicated.

CHOICE OF VEHICLES

The choice of a vehicle depends on the physicochemical properties of the drug, the mode of administration and the flavor. Water soluble drugs are given in aqueous or hydro alcoholic vehicles or in solid form (pills, capsules). Fat soluble drugs are given in oils or in semisolid vehicles.

In choosing vehicles for masking the unpleasant tastes of certain water soluble drugs it is often difficult to select the most advantageous medium. Most patients prefer *Syrup of Cocoa NF* and *Syrup of Raspberry NF* to disguise acids and the bitter taste of the salt of an alkaloid. *Syrup of Cinnamon NF* and *Syrup of Orange USP* are best for disguising the nauseating tastes of ammonium chloride, ferric and ammonium citrate, sodium salicylate and sodium bromide. The best of all purpose flavoring agents are *Syrup of Cocoa NF*, *Syrup of Prepared Cacao*, *Syrup of Raspberry NF* and *Aromatic Syrup of Erodactyon NF* in the order named.

VEHICLES AND SPEED OF ABSORPTION

Vehicles are often used to retard absorption. Drugs may be injected intramuscularly in oily suspension to prevent rapid absorption (epinephrine in oil, pitressin in oil) for rapid action an aqueous solution is given orally or parenterally. Solid preparations are absorbed slowly when implanted (pellets of steroid hormones); absorption in the intestine may fail when pellets fail to disintegrate.

Though many decry the waning of the art of pharmacy particularly in regard to coloring and flavoring there is no doubt but that this accomplishment belongs to the rococo period when the form was emphasized and the substance of relatively slight importance. Potent pharmacological agents like good food require a minimum of garnishing. The tablets of the sulfonamides are white and tasteless.

As a matter of fact there are certain patients who measure the potency of the preparation by its unpleasant taste and bad odor. The tincture of valerian and asafoetida in the combination that is euphemistically known

CHAPTER 17

PHARMACOTHERAPY VEHICLES, DRUGS APPLIED LOCAL AND TOPICAL EFFECTS

VEHICLES

A VEHICLE is an inert substance which is used as a solvent or diluent of the principal drug. When used with solid preparations (pills, capsules, powders) the vehicle (starch or lactose) facilitates administration by increasing the bulk of the medicine. Vehicles for liquid preparations taken by mouth dissolve the drug and make it palatable. Semisolid vehicles used in ointments often allow the active principle to penetrate and serve as protectives and emollients.

COMMON VEHICLES

Liquid Vehicles

1 Aromatic Waters

- Anise Water USP
- Cinnamon Water USP
- Peppermint Water USP
- Spearmint Water USP
- Bitter Almond Water USP
- Camphor Water U.S.I.

2 Syrups

- Simple Syrup USP
- Syrup of Acacia NF
- Syrup of Cinnamon NF
- Syrup of Cherry NF
- Syrup of Citric Acid USP
- Syrup of Cocoa NF
- Syrup of Prepared Cacao NF
- Syrup of Glycyrrhiza NF
- Syrup of Orange USP
- Syrup of Raspberry NF
- Syrup of Balsam USP
- Syrup of Wild Cherry USP
- Aromatic Syrup of Eriogonin NF

3 Mucilages

- Acacia USP
- Tragacanth USP

4 Hydro-alcoholic Vehicles

- Aromatic Elixir USP
- Red Aromatic Elixir NF
- Elixir of Glycyrrhiza USP
- Iso-alcoholic Elixir NF

5 Alcoholic Vehicles

Tinctures

- Tincture of Culbear NF
- Compound Tincture of Cardamom USP
- Tincture of Sweet Orange Peel USP
- Tincture of Bitter Orange Peel USP

Tincture of Cinnamon NF

Spirits

- Spirit of Peppermint USP
- Spirit of Lavender USP
- Aromatic Spirit of Ammonia USP

Semisolid Vehicles

- 1 Petrolatum
- 2 Collodion
- 3 Benzoinated Lard
- 4 Lanolin
- 5 Simple Ointment
- 6 Rose Water Ointment
- 7 Oil of Theobromine
- 8 Aquaphor

Solid Vehicles

- Talcum
- Starch
- Fuller's earth
- Kaolin
- Chalk
- Lactose

Aromatic Waters—Aromatic waters are aqueous solutions of volatile oil which may be used as vehicles for water soluble drugs. They are relatively ineffective for masking disagreeable tastes.

CHAPTER 178

PHARMACOTHERAPY ENDOGENOUS AGENTS USED FOR SYSTEMIC EFFECTS

Water the Anions and the Cations
Foodstuffs Metabolites and Vitamins
Hormones
The Therapeutic Gases
Oxygen
Carbon Dioxide
Helium

WATER, salts, minerals, fats, dextrose, proteins, vitamins, hormones, oxygen, and carbon dioxide are normal constituents of the human body which possess pharmacodynamic potentialities. They have specific efficacy when used for replacement therapy in the deficiency states as particularly illustrated by the vitamins (p 3824) and the products of the internal secretory glands (p 3825). Additionally, each of these chemical entities is capable of producing alterations beyond those in the realm of replacement therapy as best noted by *epinephrine in asthma* and by *posterior pituitary extract in the prevention and treatment of postoperative ileus*.

TABLE 10—THE PHARMACOLOGY AND THERAPEUTICS OF WATER, THE ANIONS AND THE CATIONS

Water

Aids flow of saliva; increases flow of gastric and pancreatic juices; prevents inspissation of stool; diminishes bacterial putrefaction; prevents and corrects dehydration; acts as diaphoretic and diuretic; prevents urinary precipitation of crystals and calculi.

Administer water orally by hypodermoclysis (p 3772) or by intravenous drip to maintain a urinary output in excess of 1500 cc.

Sodium Chloride

Required by those who lose fluid by sweating, emesis, diarrhea, hemorrhage or glycosuria; used by workers in "heavy" trades; prevents and relieves crises of adrenal cortical deficiency.

Give Sodium Chloride U.S.P. as capsules or compressed tablets of table salt or as intravenous drip of physiologic saline or Ringer's solution.

Ammonium Chloride

For production of the apertic acidosis; to aid in diuresis; relieve premenstrual tension (p 2456) and as urinary antiseptic (p 2456).

For therapeutic acidosis in premenstrual tension and edema, order Ammonium Chloride U.S.P. in compressed tablets of 0.5 gm. (~14 grains). Give 6 to 16 tablets (3-8 gm. [45 to 120 grains]) daily for 2 or 3 days on and 1 or 2 days off. As expectorant, use *Liquor Ammoniae Aromaticus* N.F. 2 cc. in $\frac{1}{4}$ glass of water every 2 or 3 hours.

Potassium

To prevent and treat attacks of familial periodic paralysis (p 1416); in Meniere's disease (p 1448) and allergies as feeble diuretic.

Potassium Chloride U.S.P. is given orally in 0.5 gm. (7½ grains) tablets or capsules. Prescribe 3-10 gm. (~45 to 150 grains) daily for rapid action; inject 50 cc. of 2 per

as 'bums' mixture' may be highly recommended for gustatory and olfactory offensiveness

DRUGS APPLIED FOR LOCAL AND TOPICAL EFFECTS

Local and topical applications aim to modify the function or structure of the treated tissue. With few exceptions significant systemic absorption

TABLE 209 —LOCAL AND TOPICAL PHARMACOTHERAPY

| | |
|---------------------|--|
| Tegumentary System | Cosmetics anesthetics analgesics anodynes astringents, cleansers agents deodorants emollients demulcents lubricants, protective protein precipitants rubefacients antidotes antipruritics decontaminants depilatories diaphoretics keratolytics keratoplastics parasiticides bactericides fungicides venereal prophylactics See p 311 |
| Digestive System | Cosmetics mouth washes dentifrices acids ferments, antacids absorbents emetics protectives antemetics lubricants, increasers of stool bulk alterers of stool bacteria, irritants, cholagogues laxatives and cathartics anthelmintics bactericides See p 166 |
| Eye | Cosmetics collyria anesthetics mydriatics miotics astringents, protectives bactericides See p 1343 |
| Respiratory System | Vasoconstrictors emollients bronchodilators inhalant bactericides See p 202 |
| Reproductive System | Aphrodisiacs contraceptives venereal prophylactics bactericides trichomonocides See p 2512 |

is not anticipated except as an accident or an idiosyncrasy. Indeed appreciable absorption may defeat the purpose of the applied substance since local concentration falls and distant undesirable side effects may be produced.

Urea

As diuretic in circulatory edema (p 841)

Daily dose of 50-100 gm Give iced in watery saturated solution

Amino acids

Sterile solutions commercially available for intravenous drip to meet nitrogen requirements

See also Antihistamine drugs (p 505)

Vitamins A and D

Specific in rickets and infantile tetany (p 2778) to prevent infection and dental caries to relieve osteoarthritis and assist in healing of fractures

Richest supply in percomorph liver oil (1 gm \approx 60,000 units of A and 8500 units of D)

Thiamine Chloride

Specific in beriberi (p 699) May be indicated in neuritis especially in tabetic crises hyperthyroidism and diabetes mellitus

Give intravenously in doses of at least 50-100 mg (solutions available 1 cc \approx 100 mg)

Riboflavin

Specific in ariboflavinosis (p 623) Try in keratitis and other chronic lesions of the eye

Give intravenously in 1 or 2 mg doses (1 cc \approx 0.5 mg)

Niacin

Specific in pellagra (p 679) to prevent toxicity of sulfonamide and in radiation therapy

Give orally in 25-100 mg doses for intravenous use (1 cc \approx 10 mg or 50 mg)

Ascorbic Acid (Vitamin C)

Specific in scurvy (p 2837) Try in hemorrhagic states pulmonary tuberculosis pertussis and rheumatic fever to prevent drug toxicity in arsenotherapy and in treatment with sulfonamides

Give intravenously in 50-100 mg doses Ampoules available (1 cc \approx 50 mg)

Alpha-tocopherol (Vitamin E)

Sometimes indicated in sterility threatened abortion muscle dystrophies and amyotrophic lateral sclerosis (p 1503)

Inject 50 mg intramuscularly

Vitamin K

Sometimes indicated in hemorrhagic conditions and obstructive jaundice in pregnancy in last month to prevent hemorrhagic diseases of newborn (p 2789)

Prescribable salts orally and inject 1-2 mg of Menadione USP intramuscularly

TABLE 212—THE PHARMACOLOGY AND THERAPEUTICS OF THE HORMONES

Thyroid Extract USP

Specific in myxedema cretinism and hypothyroidism (p 1191) Sometimes indicated in obesity sterility and habitual abortion Diuretic in nephrosis (p 2389)

Regulate dose by basal metabolic rate Use enteric-coated tablets USP (p 1189) Thyroidin USP is more expensive and has no superiority except for intravenous use ; dose of 2 or 5 mg See also Antithyroid preparations (p 1211)

Parathyroid Extract USP

Specific in adult tetany (p 723) Sometimes indicated in lead poisoning and for prevention of radiation sickness

Recommended only in parathyroid tetany Use Parathyroid Extract USP of which 1 cc \approx 100 units (p 726)

cent solution intravenously Use great caution Avoid entirely in adrenal cortical and renal insufficiency

Calcium

As coagulant to enhance digitalis action in tuberculosis to combat exudation in effusions to prevent and relieve low calcium tetany as antispasmodic in gallbladder and urinary colic insoluble salts in tooth powders and pastes (p 166^o)

For systemic effect use slow intravenous injection of 5 or 10 per cent Calcium Chloride USP Do not exceed 20 cc of 5 per cent or 10 cc of 10 per cent solution For intramuscular injection give 5 or 10 cc of Calcium Gluconate USP

Magnesium

Antacid and absorbent saline laxative analgesic wet dressing antispasmodic anticonvulsant analgesic and sedative

For antacid and absorbent give Magnesium Oxide Magnesium Carbonate or Magnesium Trisilicate USP Epsom salts (Magnesium Sulfate) USP may be used for wet dressings and as a saline laxative In duodenal drainage instill 25 per cent solution for intramuscular injection in treatment of convulsions cautiously employ 2 to 4 cc of 25 per cent magnesium sulfate

Ammonium

Reflex circulatory stimulant

Use smelling salts (Aromatic Spirits of Ammonia USP)

Phosphates and Phosphites

Antacid alkalinize urine saline laxative nerve tonic

As antacid use Calcium Phosphate USP For alkalinization use Calcium Dihydrogen Phosphate USP For saline laxative prescribe Sodium Phosphate USP Hypophosphites are valueless as "nerve tonics"

Sulfates

Saline laxatives

Use Magnesium or Sodium Sulfate USP for saline laxative effects

Iodides

In tertiary syphilis (p 3286) thyroid hyperplasia (goiter) (p 1103) hyperthyroidism (p 1197) as expectorant as mycocide

Iodide action obtained from any of salts Most popular are saturated Potassium Iodide USP and Compound Solution of Iodine (Lugol's) Either is efficacious in tertiary syphilis prevention and treatment of simple goiter and control of hyperthyroidism Less useful as mycocide in actinomycosis (p 489) and objectionable as an expectorant

Radioactive Phosphorus

Use P-32 in leukemias (p 1100) and polycythemia (p 109^o)

If P-32 can be obtained results equal those of roentgentherapy in leukemia and polycythemia vera

TABLE 111—THE PHARMACOLOGY AND THERAPEUTICS OF FOODSTUFFS METABOLITES AND VITAMINS

Dextrose

To combat acidosis protect liver decrease intracranial tension correct hypoglycemia Give intravenously as 20-50 cc of 50 per cent Dextrose USP or Sucrose (orbsitol) USP

Lactose

To alter intestinal flora in "auto-intoxication"

Prescribe as beta lactose (15-30 gm) with acidophilus milk

OXYGEN

The tissues require a constant supply of oxygen. If the oxygen supply is wanting or strikingly deficient, life cannot be maintained for more than a few moments.

Atmospheric air at sea level normally contains 20.5 per cent of oxygen. With this concentration the hemoglobin of the blood is approximately 95 per cent saturated, all else being equal. Anoxemia may result when the atmospheric air contains lesser amounts of oxygen, as at levels above 10,000 feet, and when pathologic conditions result in diminution of the oxygen content of the blood and tissues.

Types of Anoxia—Anoxia is encountered as histotoxic, anemic, stagnant, and anoxic varieties. In *histotoxic anoxia*, oxidative mechanisms of the tissues are paralyzed, as in cyanide poisoning. In the *anemic forms*, the oxygen impoverishment is due to a reduction in the amount of circulating hemoglobin, as in chronic anemia or acute blood loss due to hemorrhage. The *stagnant* type of anoxia results from a slowing of the circulation and a delivery of oxygen at an inefficiently low pressure, as commonly encountered in circulatory failure, shock, and in obstruction to venous return. The *anoxic type* of anoxia results from the alteration of the gaseous metabolism by the low oxygen tension in the inspired air. This variety, which responds best to oxygen therapy, is encountered in aviation at high altitudes and with abnormalities of the cardiopulmonary mechanisms.

Clinical Manifestations of Anoxia—The clinical manifestations of anoxemia include headache, nervousness, nausea, vomiting, restlessness, mental confusion, and increased rapidity of the pulse rate. With more marked deficiency, the complaints increase quantitatively, and dyspnea, delirium, ashen cyanosis, or the pallor of shock are noted.

The Administration of Oxygen—Oxygen is a colorless, odorless, and tasteless gas. It is marketed in compressed form in sealed cylinders, which are equipped with reducing valves for delivery of the gas, and a calibrated flow regulator. Most regulators are equipped with gauges showing the amount of oxygen in the tank and the flow in liters per minute. Medicinal oxygen differs from commercial oxygen only in that it is more expensive. The practitioner should acquaint himself with the nearest commercial source for oxygen cylinders; tanks are often available for emergency use at the local police or fire departments.

Oxygen may be administered to the patient through the nasal catheter or the face mask and in oxygen tents or chambers.

Nasal Catheter Technique—For oxygen administration by the nasal catheter, the nostril is anesthetized with a cocaine substitute (2 per cent nupercaine or metycaine), and a lubricated, soft rubber tube is inserted into the nasopharynx but without impingement on the posterior wall. The free end of the tube is attached to a wash bottle containing a two-way stopper, which is also joined to a length of tubing passing to the reducing valve and cylinder. Using a flow of 5 liters per minute, the patient will ordinarily obtain approximately 37 per cent of oxygen; at a flow of 8 liters, a 40 per cent concentration may be achieved. The nasal catheter method of oxygen administration is highly successful unless the patient is unusually sensitive and cannot stand the discomfort of the nasal tubes.

Insulin USP

Specific in diabetes mellitus (p 1246) Sometimes indicated in debilitated states and to combat anorexia as in tuberculosis To produce hypoglycemic convulsions in psychoses (p 1329)

Use soluble Insulin USP or Protamine Zinc Insulin NNR subcutaneously with safeguards against hypoglycemic reactions (p 1243)

Estrogen USP

Specific in hypo-ovarian states and menopause (p 2493) Use in treatment of gonorrheal vulvovaginitis of children kraurosis vulvae and atrophic rhinitis to control metastases in prostatic carcinoma (p 2460) May try in diabetes mellitus diabetes insipidus and senile osteoporosis to inhibit and suppress lactation (p 2719)

Give synthetic diethylstilbestrol orally Also available for use in ointment vaginal suppository sublingual medication or intramuscular injection Crystalline and noncrystalline estrogens also official (p 2525)

Progestin NNR

Has been used in threatened abortion habitual abortion dysmenorrhea and functional bleeding

For intramuscular injection (p 2517)

Androgen

Specific in eunuchism and male climacteric (p 2401) may prevent and relieve prostatic hypertrophy to control metastases in carcinoma of breast (p 2520) for relief of menorrhagia and dysmenorrhea in pituitary basophilism and dwarfism may inhibit or suppress menstruation and lactation

Available in ointment sublingual medications suppositories and for intramuscular injection

Adrenal Cortex Extract NNR

Specific in adrenal cortical deficiency and Addison's Disease (p 1267) May prevent and relieve shock asthenic states and hypotension

Desoxycorticosterone acetate for intramuscular use and subcutaneous implants (p 1271)

Adrenal Cortex Extract NNR (1 cc = 50 dog units)

Posterior Pituitary Extract

Specific in diabetes insipidus (p 1180) to stimulate uterine and intestinal muscle in herpes zoster in ureteral calculi

Solution of Posterior Pituitary USP contains all principles Pitresin NNR has mostly pressor fraction and Pitocin NNR is essentially oxytocic pitressin tannate in oil by intramuscular injection is best for diabetes insipidus

Anterior Pituitary Growth Promoting Hormone

In dwarfism and Simmonds disease (p 1169)

No official preparation but Antuitrin Growth is reliable

Anterior Pituitary Gonadotropic Hormones

In hypogonadism infantilism cryptorchidism ovarian deficiency and menorrhagia

Official preparation is Chorionic Gonadotropin NNR

Liver Extract USP and Folic Acid

Specific in primary hyperchromic anemia (p 1077)

THE THERAPEUTIC GASES

Oxygen carbon dioxide and helium alone or in combination may be administered for therapeutic purposes

carbon monoxide morphine and barbiturate poisonings in acute alcoholism following general anesthesia to control intractable hiccup in pulmonary atelectasis shock and the asphyxia of the new born When a therapeutic hypercapnia is produced the gas is given intermittently for two or three minutes in concentrations of 5 to 10 per cent

HELIUM

Helium has been introduced into medicine for the relief of conditions in which respiration is difficult due to mechanical obstruction Mixtures

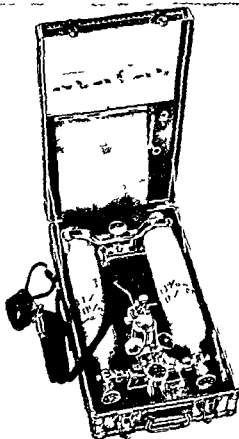


Fig 112.—Helium inhaler for the administration of carbon dioxide-oxygen mixtures

containing 80 per cent helium and 20 per cent oxygen require only half the suction pressure that is needed to breathe ordinary air

Helium gas may be purchased in cylinders with governmental authority It is administered in a helium proof oxygen tent or any closed circuit rebreathing apparatus The use of helium is an institutional procedure which requires expert observation It may be tried as an emergency measure in *obstructive dyspnea status asthmaticus* and the acute pulmonary edema that follows *industrial gas poisonings*

action increases cardiac filling and output slows the heart rate and causes peripheral vasodilatation

Hypercapnia—Inhalation of an excess of carbon dioxide may within a few moments cause distressing hypercapnia. If continued the patient develops acidosis, convulsions, unconsciousness, coma and respiratory failure which may prove *fatal*.

Acapnia—A low tension of carbon dioxide may result in alkalosis and respiratory failure. The acapnic state is most easily produced by hyperventilation when the forced breathing eliminates carbon dioxide in excessive amounts. The indicated therapy of the asphyxial states associated

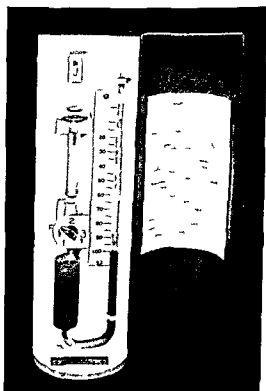


Fig. 1121—Oxygen analyzer. The photograph does not show the rubber tubing which attaches to this equipment and may be inserted into the tent. Oxygen concentration within the tent should be maintained at 40 to 60 per cent. Oxygen content within the tent is in some hospitals analyzed every four hours by the intern, the nurse or by a technician.

with acapnia is the administration of carbon dioxide with or without oxygen.

The Administration of Carbon Dioxide—Carbon dioxide is marketed under pressure in steel cylinders. The gas is introduced by means of the mask. When administered alone it is given in concentrations of 5 to 10 per cent. Cylinders containing a combination of oxygen and carbon dioxide are available; the usual ratios are 5 or 10 per cent CO_2 and 95 or 90 per cent of oxygen.

Therapeutics—Carbon dioxide is administered therapeutically for the relief of acapnia, in anoxia with respiratory failure, for the treatment of

acid acetanilid and antipyrine A single tablet may cause generalized urticaria edema of the face tongue or larynx or cyanosis or circulatory

TABLE 214—PHARMACOLOGY AND THERAPEUTICS OF ANALGESIC ANTIPIRETTICS

Temperature

Depression of heat regulating center in hypothalamus

Ant pyretic action is exhibited only in febrile

Pain

Depression of pain threshold in optic thalamus

Analgesic effect is best noted in relief of constant, dull pain

Skin

Oil of Wintergreen is counterirritant

Circulation

No evidence of depression

Analgesic antipyretics are not circulatory depressants they do not "weaken the heart" they are not contraindicated in those who claim to have "weak hearts" the reputation for cardiac depression is based on the cyanosis due to methemoglobinemia

Digestion

Gastric irritation may cause nausea and vomiting administer with bicarbonate and charged water

Bile

Salicylates are cholagogue may be tried in biliary dyskinesia (p 2807)

Liver

Cinchophens may cause fatal hepatitis (p 1963)

Urinary Acid

Salicylates and cinchophen increase urinary excretion of uric acid but colchicine affords greatest relief in gout (p 2807)

Joint Fluids

No specific localization in arthritis

Kidney

80 per cent excretion in urine which may show evidence of renal irritation (albuminuria and hematuria)

Blood

Acetanilid may produce methemoglobinemia and sulfhemoglobinemia aminopyrine may produce agranulocytosis (p 1096)

Infectious Organisms

Quinine is specific in malaria (p 507) salicylates widely used in rheumatic fever (p 186)

Uterus

Coal tar relieves dysmenorrhea (p 2561)

Many popular female remedies are tablets whose main ingredient is an analgesic antipyretic

collapse akin to shock Less alarming are the minor nuisances of nausea vomiting and toxic eruptions

Overdosage results in syndromes simulating cinchonism (p 862) and salicylism (p 198) There may be nausea vomiting albuminuria hema

Prescription 3

| | |
|------------------------|------|
| R Acetylsalicylic Acid | 4.5 |
| Codeine Phosphate | 0.25 |
| Make 15 capsules | |

Prescription 4

| | |
|-------------------------------|-----|
| R Acetylsalicylic Acid | 9.5 |
| Codeine Phosphate | 0.5 |
| Make 15 rectal suppositories. | |

The combination of an analgesic and a narcotic is recommended highly Prescription number 3 may be given orally number 4 by rectum

Prescription 5

| | |
|------------------------|------|
| R Acetylsalicylic Acid | 2.5 |
| Acetophenetidin | 2.25 |
| Phenobarbital | 0.25 |
| Make 15 capsules | |

The use of an analgesic antipyretic and a barbiturate affords considerable relief from pain associated with nervousness or restlessness (the nervous headache)

Prescription 6

| | |
|-------------------|-----|
| R Acetophenetidin | 2.5 |
| Phenylsalicylate | 2.5 |
| Make 15 capsules | |

This is the phenacetin and salol capsule once widely employed for the relief of headache of intestinal origin The salol was presumed to be an intestinal antiseptic

Administration Absorption and Excretion of Analgesic Antipyretics—The analgesic antipyretics are usually given orally and are well absorbed from the upper bowel They produce gastric irritation to greater or lesser degree Following the ingestion of a tablet of aspirin gastroscopy reveals intense local congestion and exudation Clinically this observation is confirmed by the large number of patients who develop vomiting after the use of some one or all of these products The local gastric reaction is minimized by using an effervescent product well diluted after meals if a tablet or a powder is prescribed a heaping teaspoonful of bicarbonate of soda and a full glass of water should accompany the medication

If oral administration is unsuccessful a suppository may be substituted or the soluble sodium salicylate may be instilled in an enema of at least 60 cc of starch water The intravenous injection of sodium salicylate suggested in rheumatic fever (p 186) is not without hazard and is not endorsed as a routine procedure

The salicylates are rapidly distributed and may be identified in tissue fluids a few moments after administration They do not exhibit any elective localization in joint fluids Approximately 20 per cent of the dose is destroyed in the body but the remainder is excreted especially by the kidneys Urinary excretion causes irritation as manifested by frequent albuminuria and hematuria (p 2306) A pseudoreduction of Benedict's solution also is observed

Toxicology—The analgesic antipyretics frequently produce untoward manifestations *Idiosyncrasy* is common as illustrated by the granulopenic effect of aminopyrine and the hepatotoxic action of the cinchophens Allergy like reactions in the sensitive may follow the use of acetylsalicylic

TABLE 215.—PREPARATIONS AND DOSAGES OF SEDATIVES AND HYPNOTICS (Continued)

| | |
|--|---|
| Barbital Sodium (Medinal) | 1 rounder tablet 0.3 gm (5 gr) Suppository 0.6 gm (10 gr) |
| Dial N N R | Tablet 0.1 and 0.3 gm ($1\frac{1}{2}$ –5 gr) Ampoule 0.1 gm ($1\frac{1}{4}$ gr) |
| Ipral Calcium N N R | Tablet 0.01 and 0.12 gm (γ_3 and 2 gr) |
| Ipral Sodium N N R | Tablet, 0.24 gm (4 gr) Elixir 4 cc = 0.06 gm (1 gr) |
| Neonal N N R | Tablet 0.09 gm ($1\frac{1}{2}$ gr) |
| Nostal N N R | Tablet 0.09 gm ($1\frac{1}{2}$ gr) |
| Ortal Sodium N N R | Capsule 0.18 and 0.3 gm (3 and 5 gr) |
| Pentobarbital Sodium USP (Nembutal) | Capsule 0.21 and 0.1 gm (γ_3 and $1\frac{1}{2}$ gr) Suppository 0.12 gm (2 gr) Ampoule 0.5 gm ($7\frac{1}{4}$ gr) |
| Pentothal Sodium N N R | Ampoule 0.5 and 1.6 gm ($7\frac{1}{2}$ –15 gr) Pentothal sodium is the preparation of choice for intravenous anesthesia (p 3923) |
| Pernoston N N R | Tablet, 0.18 gm (3 gr) |
| Phanodorm N N R | Tablet 0.18 gm (3 gr) |
| Phenobarbital USP (luminal) | Tablet, 0.015 0.03 and 0.09 gm ($\frac{1}{4}$ $\frac{1}{2}$ and $1\frac{1}{4}$ gr) Elixir 4 cc = 0.015 gm ($\frac{1}{4}$ gr) Phenobarbital and dilantin sodium are most effective in the treatment of epilepsy (p 1515) Dilantin may cause toxic symptoms (p 3845) |
| Phenobarbital Sodium | Ampoule 0.1 ^o and 0.3 gm (2 and 5 gr) |
| Sandoptal N N R | Tablet 0.19 gm (3 gr) |
| Secunal Sodium N N R | Capsule 0.0 and 0.1 gm ($\frac{1}{4}$ and $1\frac{1}{2}$ gr) |
| Paraaldehyde USP | 4–30 cc Paraldehyde has an evil taste which is imperfectly disguised by prescription in beer or a cola drink |
| Dilantin Sodium N N R | Capsule 0.09–0.09 gm ($\frac{1}{4}$ and $1\frac{1}{4}$ gr) Phenobarbital and dilantin sodium are most effective in the treatment of epilepsy (p 1515) Dilantin may cause toxic symptoms (p 3845) Dilantin is too toxic for its therapeutic potentials |
| Nervanol | |
| Sulfonmethane (Sulfonal) N F | Powder 0.5–1.0 gm ($7\frac{1}{2}$ –15 gr) |
| Sulfonethylmethane (Trional) | Powder 0.5–1.0 gm ($7\frac{1}{2}$ –15 gr) |
| Sulfonethylmethane (Tetronal) | Powder 0.5–1.0 gm ($7\frac{1}{2}$ –15 gr) |
| Urethane | Powder 1.0 gm (15 gr) Sulfonal, tetronal and urethane are weak and slowly excreted. They have been replaced by the barbiturates |
| Thioamethanol USP (Avertin with Amylene Hydrate N N R) | Solution of 0.5 per cent 0.08 cc per kg of body weight, of freshly prepared titrated solution given rectally Avertin is used for basal anesthesia. The freshly prepared solution must be tested with Congo red indicator just before rectal instillation. The total dose should not exceed 5–8 cc for women or 8–10 cc for men. We have abandoned its use in favor of oral or intravenous barbiturate which is simpler, safer and less expensive |

tumor dizziness ringing in the ears tinnitus head noises confusion restlessness mania or hallucinations

Cumulative poisoning is particularly encountered in patients with chronic headache who resort to the habitual use of *acetanilid*. The symptoms include anorexia nervousness irritability insomnia recurrent headache and cyanosis due to met or sulf hemoglobinemia

THE SEDATIVES AND HYPNOTICS

Sedation and hypnosis are quantitative manifestations of depression of the central nervous system. Sedatives blunt the sensitivity of the nervous tissue without the production of sleep. Hypnotics induce somnolence.

Most sedative and hypnotic drugs are not analgesic. When restlessness or insomnia is produced by pain the practitioner must resort to an analgesic antipyretic an opiate or alcohol with which the sedative or hypnotic may be effectually combined.

CHLORAL HYDRATE U S I

Chloral hydrate may be prescribed in the compressed tablet or in an aqueous solution. It can be administered by rectum in a small starch enema. Absorption is rapid and complete by either route within a few moments.

Therapeutics—In the laboratory chloral hydrate is the standard drug for the study of depressant action. In practice it is less popular than the more widely advertised barbiturates. This is unfortunate since chloral produces a normal type of sleep; it is rarely followed by a "hangover"; idiosyncrasies are most unusual and rashes have never been encountered in our experience.

TABLE 215—PREPARATIONS AND DOSAGES OF SEDATIVES AND HYPNOTICS

| | | | | | | | | |
|--|---|-------------------|----|----------------------|-----|------------------------------|------|-----------------------------|
| Chloral Hydrate U S P | Tablet 0.3-1 gm (5-15 gr) | | | | | | | |
| | Chloral and alcohol form "knockout drops" | | | | | | | |
| | A popular prescription to replace the bromide and chloral mixture of the N F is | | | | | | | |
| | <table> <tr> <td>℞ Chloral Hydrate</td><td>50</td></tr> <tr> <td>Sodium Phenobarbital</td><td>0.5</td></tr> <tr> <td>Syrup of Orange Peel to make</td><td>60.0</td></tr> <tr> <td>1 teaspoonful every 3 hours</td><td></td></tr> </table> | ℞ Chloral Hydrate | 50 | Sodium Phenobarbital | 0.5 | Syrup of Orange Peel to make | 60.0 | 1 teaspoonful every 3 hours |
| ℞ Chloral Hydrate | 50 | | | | | | | |
| Sodium Phenobarbital | 0.5 | | | | | | | |
| Syrup of Orange Peel to make | 60.0 | | | | | | | |
| 1 teaspoonful every 3 hours | | | | | | | | |
| Chlorobutanol (Chloretone) N N R Sodium Bromide U S P | Capsule 0.3 gm (5 gr) | | | | | | | |
| | Tablet 1-5 gm (15-75 gr) | | | | | | | |
| | Sodium bromide is best prescribed in the effervescent tablets but bromism (p 3839) must be guarded against | | | | | | | |
| | | | | | | | | |
| Barbiturates | Tablet 0.06 gm (1 gr) | | | | | | | |
| | Capsule 0.25 gm (4 gr) | | | | | | | |
| | Capsule 0.06 and 0.18 gm (1 & 3 gr) | | | | | | | |
| | Fluxir 4 cc = 0.015 gm ($\frac{3}{4}$ gr) | | | | | | | |
| | Elixir 4 cc = 0.03 gm ($\frac{3}{4}$ gr) | | | | | | | |
| | Capsule 0.06 and 0.18 gm (1-3 gr) | | | | | | | |
| | Suppository 0.18 gm (3 gr) | | | | | | | |
| | Ampoule 0.24 0.5 and 1.0 gm (4 gr and 15 gr) | | | | | | | |
| | Powder pill 0.3 gm (5 gr) | | | | | | | |
| | Barbital is habit forming and should be avoided | | | | | | | |
| Barbital (Veronal) U S I | | | | | | | | |

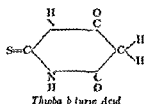
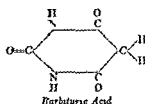
BARBITURATES

The barbiturates are a useful and reliable group of depressants of the central nervous system. Since the introduction of the first compound *barbital* in 1903 several hundred closely related substances have been synthesized and subjected to pharmacological study. Many of these have been used clinically with gratifying results.

The barbiturates possess a basic ring substance to which a variety of chemical groups have been added to produce differences in activity. The individual drugs vary in the intensity of action, the rapidity of onset and duration. Such differences widen the therapeutic usefulness of the entire group but require a thorough understanding of the individual members are to be employed with the maximum effectiveness.

The barbiturates are used for sedation, hypnosis, pre-anesthetic preparation, basal and general anesthesia and for the treatment of convulsive diseases and poisonings.

Chemical Relationships.—The parent compounds are *barbituric acid* and *thiobarbituric acid*, condensation products of urea and thiourea with malonic acid. Most barbiturates in use today belong to the barbituric acid series. The only thiobarbiturate which has become popular is *pentothal*.



The replacement of hydrogen by *aliphatic*, *aromatic* or *heterocyclic* radicals confers narcotic powers on the inactive ring structures. Potency, efficiency and toxicity vary with the chemical configuration. Two short *aliphatic* chains give long acting drugs (*barbital*, *phenobarbital*). One short and one long chain yield short acting *pentobarbital*, *sodium*. Two long chains produce ineffective compounds.

The potency of the individual compounds increases with the weight of the molecule. Activity is enhanced by the placing of halogens on the *aliphatic* groups (*pernoston*).

The free acids are only sparingly soluble in water, a property which delays the onset of action. The substitution of *sodium* for one of the hydrogens of the *NH* groups yields freely soluble compounds whose action is prompt. These can be given intravenously when necessary (*sodium amytal*, *sodium pentobarbital*, *pentothal*, *sodium*, *sodium phenobarbital*).

BARBITURATE PRESCRIPTIONS

Prescription 1

| | |
|---|------------|
| Sodium Phenobarbital | 0.5 |
| Pepsin | 10 |
| Charcoal | 60 |
| Oil of Peppermint | 10 |
| Glycerin | 200 |
| Water | qs ad 1200 |
| Shake Well Label | |
| Take 1 teaspoonful after meals and at bedtime | |

The unpopularity of chloral is due partially to its reputation for forming knockout drops with alcohol and its alleged depressant action on the heart. Each of these hazards is exaggerated. The synergism with alcohol in point of fact may be employed therapeutically in maniacal patients. The so called cardiac depression is of some value in treating apprehensive patients with essential hypertension (p 900).

Chloral or its derivative *chlorobutanol* (*chloretone*) NNR is the principal drug in most remedies prescribed for the prevention and cure of *seasickness*.

Destruction—Chloral hydrate is rapidly and completely destroyed in the body. The mechanism of excretion is not well understood but it may cause a pseudoreduction in urine tested with Benedict's solution (p 3674).

Idiosyncrasy and Poisoning—We have never observed idiosyncrasy to chloral. Poisoning may occur with excessive dosage under which circumstance the patient is profoundly comatose, reflexes are abolished and death may occur from respiratory paralysis.

Treatment of Chloral Poisoning—Treatment requires removal of the drug from the stomach by gastric lavage. There is no specific antidote. Body heat is maintained and respiration is artificially induced by a pulmotor if necessary.

THE BROMIDES

Sodium bromide possesses all the virtues and faults of the more expensive sodium calcium potassium, ammonium and strontium salts. Each is readily absorbed from the digestive tract and circulates in high concentration in the serum. Tissue retention is excessive and excretion occurs slowly in the urine.

Pharmacology and Therapeutics—The bromides depress the central nervous system and affect principally the psychic functions and the reflexes. They are used to relieve nervous irritability to produce sleep for the prevention and treatment of the convulsive phenomena of epilepsy (p 1515) as a seasickness remedy and in the control of hyperthyroidism.

We have abandoned the use of the bromides because of the dangers of bromism (p 3840). The barbiturates are substituted as sedatives, hypnotics and anticonvulsants. Dilantin is more efficacious in epilepsy and the iodides are specific in hyperthyroidism (p 1197).

Bromism—For effective therapeutic action an adequate concentration of the bromide ion must be built up in the tissues and body fluids. As a result the symptoms of bromism are seen with great frequency. The bromide eruptions are often pustular and occur most objectionably on the face. Patients with acne (p 3358) or a tendency to acne are particularly subject to an accentuation of the dermatosis if they are given a bromide preparation. The undesirable systemic effects of bromism include chronic depression, anorexia, cachexia, sexual impotence, gastro-intestinal disturbance, psychic deterioration, confusion, hallucination, disturbances in speech and gait as well as nutritional defects. Psychiatrists who engage in institutional work report that many applicants for admission suffer more from bromide intoxication than the original illness for which the drug was taken.

The treatment of bromism consists in discontinuance of the drug and the daily administration of 6 to 9 gm (90 to 135 grains) of sodium chloride with at least 4000 cc of fluid.

In patients with hepatic or renal insufficiency the action of barbiturates is intensified and prolonged since the drugs are neither destroyed nor

TABLE 218—PHARMACOLOGY AND THERAPEUTICS OF THE BARBITURATES

| Effect | Preparation of Choice | Comment |
|--------------------------------------|---|---|
| Depression of central nervous system | Phenobarbital 15 mg ($\frac{1}{2}$ gr) Mistura Nigra (p 3340) See also Barbiturates in Table 215 p 3836 | Occasional idiosyncrasy and dermatitis Mistura Nigra is particularly useful in the management of psychosomatic disorders (p 1541) Often it can be prescribed in the manner of a therapeutic test for a period of ten days. At the end of this time many baffling symptoms as well as those which suggest organic disease may disappear to the relief of all concerned |
| Hypnosis | Seconal sodium 90 mg ($1\frac{1}{2}$ gr) Amytal sodium 180 mg (3 gr) See also Barbiturates in Table 215 p 3836 | May give half or double doses For those who have difficulty falling asleep the more soluble seconal preparation is preferable Those whose problem is early wakefulness are given the less soluble barbiturate (Amytal) |
| Analgesia | Not effective but may potentiate | coal tars as in prescription 3 (p 3340) |
| Anticonvulsant | Phenobarbital 15 mg ($\frac{1}{2}$ gr) (As a motor cortex depressant in epilepsy) | Superior to bromide inferior to dilantin (p 3844) but may be used in combination (p 1515) |
| Basal anesthesia | Seconal sodium 180-270 mg (3-4½ gr) Amytal sodium 360-540 mg (6-8 gr) See also Major Surgery p 3992 | Ideal for ease of administration and safety. Rarely produces motor excitation. May combine with morphine or demerol and scopolamine |
| Obstetrical anesthesia | Seconal sodium 180-270 mg (3-4½ gr) Amytal sodium 360-540 mg (6-8 gr) See also Obstetrics p 617 | Often causes motor excitation |
| General anesthesia | Pentothal sodium | Pentothal sodium is the preparation of choice for intravenous anesthesia. Use freshly prepared 2.5 or 5% solution with great caution as elsewhere described (p 592) |
| Cocaine antidote | Phenobarbital 1-30 mg ($\frac{1}{4}$ -½ gr) See also Cocaine p 3915 | Sedative or hypnotic dose. Use with all local anesthetics to prevent or relieve cerebral excitement and toxicity (p 3915) |

excreted efficiently. Renal disease prolongs the action of the long acting drugs while liver pathology extends that of the short acting members of the series.

Mistura nigra (black mixture) one of the most useful prescriptions that the internist or practitioner employs is of particular value in the management of various functional disorders especially those in which gastro intestinal symptoms are the chief subjective complaints (p 1767)

The original prescription for the black mixture was made with sodium bromide 15 grams to the teaspoonful For the reasons mentioned elsewhere (p 3338) sodium phenobarbital is preferable and has been substituted

Prescription 2

| | |
|------------------------|-------|
| Ephedrine Sulfate | 0.375 |
| Phenobarbital | 0.25 |
| Make 15 capsules | |
| Take one every 3 hours | |

Prescription is beneficial in the treatment of patients who have allergic disorders The barbiturates offset the cerebral stimulation of the sympathomimetic substance without interfering with its action on the exudative or spasmodic disorder (vasomotor rhinitis or asthma)

Prescription 3

| | |
|------------------------|------|
| Acetylsalicylic Acid | 4.75 |
| Phenobarbital | 0.25 |
| Make 15 capsules | |
| Take one every 3 hours | |

Prescription presents the combination of an analgesic and the sedative It is of particular value in what is termed nervous headache

Prescription 4

| | |
|---|-------------|
| Chloral Hydrate | 60 |
| Phenobarbital | 0.25 |
| Syrup Cocoa Comp | q.s ad 60.0 |
| Take 1 teaspoonful every 3 hours if necessary | |

Prescription is intended to replace the popular bromide and chloral mixture of the NF The reason for the substitution of the barbiturate for the bromide has been previously discussed (p 3338)

Detoxification Elimination—The barbiturates are absorbed well from the intestinal tract after oral or rectal administration The speed of absorption is variable and is longer with the long acting preparations (barbital phenobarbital) than with shorter acting members (seconal) Gastro intestinal absorption is enhanced by the use of soluble sodium salts

There is rapid absorption from subcutaneous and intramuscular sites of injection

In general the fate of a barbiturate in the body depends upon its chemical structure The *shorter acting compounds* whose molecular weight is high due to the presence of one long side chain (amytal), are destroyed in the liver where the malonyl urea ring is broken up The end products are excreted in the urine The *derivatives of lower molecular weight* (intermediate acting) are partly destroyed in the liver and partly excreted unchanged *Barbiturates with short alkyl side chains* (low molecular weight) are excreted unchanged in the urine The *thiobarbiturates* (pentothal) are relatively unstable and are destroyed in the liver

The duration of action depends upon the rates of destruction and excretion Renal excretion is comparatively slow and may lead to cumulative toxicity when long acting barbiturates are used for extended periods

intravenous infusion of physiologic saline solution. To this may be added 10 mg of amphetamine sulfate in 1 cc of isotonic sodium chloride repeated once or twice at half hour intervals if necessary.

If breathing is depressed artificial respiration is instituted preferably with a Drinker respirator. The administration of high concentrations of oxygen may prove beneficial. It is important to maintain an adequate airway to facilitate an optimal respiratory exchange. In profoundly depressed patients suction is used to remove accumulated secretions in the pharynx and trachea and to prevent aspiration.

Many pharmacological antidotes have been recommended and tried in the treatment of acute barbiturate poisoning. Our preference is for picrotoxin (p 3870) an intense stimulant and convulsant of the central nervous system. It is our opinion however that the drug should not be used until the less hazardous measures of resuscitation have failed. When the necessity arises its administration requires constant medical supervision since under normal circumstances a dose of 0.2 gm (3 gr) of picrotoxin may be lethal.

When the decision to administer the antidote has been made the physician may prepare a 1:1000 solution or purchase 20 cc ampoules which contain 3 mg per cc. If the latter are available an initial dose of 9 mg (3 cc) is given by slow intravenous injection. This amount may be repeated at 20 minute intervals to produce and maintain a state of motor hyperexcitability but convulsions are avoided if possible.

If the 1:1000 solution is prepared it may be best to add the antidote to the intravenous drip. The initial dose is 9 cc which may be injected slowly into the rubber tubing or placed in the reservoir with the infusate.

We oppose the use of other pharmacological antidotes we do not favor injections of caffeine strychnine the medullary convulsants or ephedrine.

Chronic Poisoning—Chronic barbiturate poisoning is frequently observed in those with *neuroses*, *psychoses* and *alcoholism*. These patients take increasing hypnotic doses at bed time for the relief of real or fancied insomnia. Manifestations of chronic barbiturate poisoning include depression diurnal somnolence ataxia dysarthria confusion disorientation imperfections in memory anorexia and loss of weight. Veronal poisoning is particularly prone to cause ataxia and speech disturbances with loss of deep reflexes simulating tabes (p 1464).

Habituatio—Although there is an impression that the barbiturates cause habituation we are among those who doubt the existence of addiction in the normal integrated individual. The average person may take sleeping medicine over periods of strain without running the hazard of habituatio in the same manner in which the evening cocktail or highball is consumed without exposure to the risk of alcoholism.

The senior author has taken sleeping medicines on and off for more than 25 years without finding it necessary to increase the dose and without any difficulty in discontinuing medication as soon as the indications have been dissipated. In his experience that is the sequence of events in the large number of patients who use hypnotics judiciously. When barbiturate habituatio or addiction is encountered a substratum of relatively profound neurosis or psychosis may be disclosed.

Toxicology—The barbiturates may cause untoward effects due to idiosyncrasy acute or chronic poisoning or habituation

Idiosyncrasy—Idiosyncrasy is fairly frequent with the various barbiturates particularly *phenobarbital*. Patients may develop urticaria, angioneurotic edema and various eruptions. The angioneurotic manifestations assume considerable gravity if they involve the larynx or the tongue. In some instances it may be necessary to scarify the larynx and perform tracheotomy (p. 3957).

Motor excitement occurs with relative frequency particularly from the administration of *larger doses* as in obstetrical anesthesia. Basal anesthesia with barbiturates during labor is apt to be unsafe outside of institutions. The practitioner protects his patient and himself if he insists upon constant vigilance during the administration of larger doses of barbiturates for basal or obstetrical anesthesia.

Acute Barbiturate Poisoning—Acute barbiturate poisoning is increasing in incidence due to the popular use of these drugs in nervous disorders and chronic alcoholism. During the past ten years barbiturate poisonings have almost doubled in New York City and are second only to carbon monoxide as a cause of death from chemical agents.

Acute poisoning results from accidental overdosage but more frequently from attempts at suicide. At one time *barbital* (veronal) was the agent most commonly responsible. Poisoning may however follow the use of any of the popular barbiturates notably *phenobarbital*, *pentobarbital*, *amytal*, *medinal* and *allonal*.

CLINICAL MANIFESTATIONS—The clinical picture is characterized by coma. The respirations are shallow irregular and later periodic; the pupils are usually constricted and only become widely dilated as a terminal event. The light reflex is present; the deep reflexes are depressed and often absent. In the advanced stages the pulse rate is rapid and blood pressure is low. Peripheral circulatory failure leads to anuria or oliguria.

Death may occur soon after the ingestion of the drug but is usually delayed for as long as a week. Death may be due to depression of the central nervous system with respiratory and vasomotor paralysis; it is often the result of a hypostatic pneumonitis or depression of heart muscle.

There is nothing characteristic about the autopsy findings. Patients who have been in coma for a long time show a bilateral diffuse bronchopneumonia and widespread capillary dilatation. Symmetrical necroses of the globus pallidus have been noted.

DIAGNOSIS—The diagnosis of barbiturate poisoning is made from the history and the recovery of the drug from the stomach washings and urine. Most barbiturates including the short acting preparations are to some extent excreted in the urine.

TREATMENT—The primary aim of therapy when the drug has been taken orally is the removal of as much as possible from the stomach. Immediate gastric lavage should be performed with care to prevent aspiration. If the drug has been given intravenously there is no possibility of recalling the dose.

In all cases steps should be taken to prevent acute circulatory collapse and to lessen the degree of central nervous system depression. The patient is kept warm and an adequate fluid intake is maintained by a continuous

intravenous infusion of physiologic saline solution. To this may be added 10 mg of amphetamine sulfate in 1 cc of isotonic sodium chloride repeated once or twice at half hour intervals if necessary.

If breathing is depressed artificial respiration is instituted preferably with a Drinker respirator. The administration of high concentrations of oxygen may prove beneficial. It is important to maintain an adequate airway to facilitate an optimal respiratory exchange. In profoundly depressed patients suction is used to remove accumulated secretions in the pharynx and trachea and to prevent aspiration.

Many pharmacological antidotes have been recommended and tried in the treatment of acute barbiturate poisoning. Our preference is for *picrotoxin* (p 3970) an intense stimulant and convulsant of the central nervous system. It is our opinion however that the drug should not be used until the less hazardous measures of resuscitation have failed. When the necessity arises its administration requires constant medical supervision since under normal circumstances a dose of 0.2 gm (3 gr) of *picrotoxin* may be lethal.

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Habituation—Although there is an impression that the barbiturates cause habituation we are among those who doubt the existence of addiction in the normal integrated individual. The average person may take sleeping medicine over periods of strain without running the hazard of habituation in the same manner in which the evening cocktail or highball is consumed without exposure to the risk of alcoholism.

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The treatment of barbiturate habituation requires psychotherapy by the consultant (p 1327) It may be necessary as in alcoholism to advise institutionalization

PARALDEHYDE

Paraldehyde is a colorless transparent liquid with a most offensive odor and taste Because of its disagreeable physical properties paraldehyde is used far less frequently than its efficacy and safety deserve

Dose and Administration—The official dose of paraldehyde in the USP is 2 cc (30 minims) This is an insufficient quantity A minimal effectual adult dose of paraldehyde is at least 4 cc (60 minims) and 15 to 30 cc ($\frac{1}{2}$ to 1 ounce) may be administered with safety

Paraldehyde is given by mouth by rectum or intravenously It is almost impossible to mask the *oral dose* It may be given iced in milk or with a carbonated drink such as beer The latter is particularly useful in the treatment of an excited alcoholic

Unless the patient is maniacal and struggling an excellent route of administration is the *rectal enema* A few ounces of a thin starch paste are prepared the dose of paraldehyde is added and the mixture is then instilled into the rectum using a funnel and catheter as with avertin

Intravenously the action of paraldehyde is prompt and effectual One cubic centimeter may be given slowly The needle is left in the vein as with sodium pentothal anesthesia More is added if the patient has not quieted down and fallen asleep within a very few moments The intravenous dose of paraldehyde almost invariably causes coughing due to the excretion of the irritant fumes through the bronchial mucous membrane

Pharmacology and Therapeutics—Paraldehyde is rapidly absorbed and excreted It produces sleep and relaxation the duration of action may be transitory or prolonged

Because of its unpleasant odor which clings to the vicinity of the patient for many hours paraldehyde is rarely used except in desperation for the control of acute toxic delirium convulsions and the manic states as particularly encountered in alcoholism Under these conditions there is nothing of comparable efficacy and safety

For intravenous anesthesia paraldehyde has not become popular Since the introduction of pentothal sodium there would seem no prospect that it ever will be employed on any large scale In obstetric anesthesia the administration of paraldehyde has been abandoned by all but a few enthusiasts

Toxicology—An occasional death from the use of large amounts of paraldehyde has been reported It has never been our experience to witness a fatality Untoward difficulties may be partially explained by the tendency of the drug to decompose into irritating aldehydes unless it is kept in a brown stoppered bottle

DILANTIN

Dilantin Sodium NNR is a proprietary name for sodium diphenyl hydantoinate It is marketed in capsules containing 50 and 100 mg ($\frac{1}{2}$ and $1\frac{1}{2}$ grains) The drug is readily absorbed from the gastro intestinal tract but its fate in the body has not been completely clarified Only

small percentages can be recovered in the excreta and effective concentrations are built up only after several days of therapy

Pharmacology—Dilantin has a specific and selective action on the motor cortex. It inhibits convulsions so that its principal therapeutic use is in the treatment of *epilepsy* (p 1515). The mode of action is not completely understood since patients may improve clinically despite persistence of cerebral dysrhythmia in the electroencephalogram.

Therapeutics—Dilantin is more effective than phenobarbital and the bromides in the treatment of epilepsy. Unlike the older drugs its anti-convulsant action is not accompanied by a sedative or depressant effect. It is useful in controlling convulsions and psychomotor equivalents but has less value in the management of petit mal. While many patients are controlled with dilantin alone others seem to do better when phenobarbital and dilantin are given alternately or in combination; the details are elaborated in the section on *Epilepsy* (p 1515).

Toxicology—Toxic reactions are frequently observed with dilantin. Under ordinary circumstances they are not sufficiently distressing to



Fig 1126—Gingivitis due to dilantin

contraindicate further therapy. Vertigo, nervousness, tremors, nystagmus, diplopia, blurring of vision, ptosis, dysarthria, drowsiness, confusion, headache, insomnia, belligerence, and a rare psychosis have been noted. Generally the untoward manifestations occur late in the first week or early in the second week of medication and they can be relieved by temporary cessation or adjustment of dosage.

Because of its alkalinity dilantin often causes gastric irritation which may be relieved by taking the capsule with or after meals. Occasionally it produces severe abdominal pain suggesting an acute surgical catastrophe. The absence of leukocytosis, rigidity, and fever aids in the differential diagnosis.

The *skin manifestations* include a mild dermatitis and eruptions which generally occur late during the second week of treatment. The majority of these are mild; the more severe tegumentary manifestations such as dermatitis exfoliativa and purpura are rarely encountered. Perhaps the most characteristic toxic effect of dilantin is the production of *gingival hyperplasia*. The gums may become sore as early as two to four weeks

after the initiation of treatment or the hyperplasia may not appear for a few months. The patient notes that the gums bleed easily from slight trauma and this is sufficient indication for temporary cessation of treatment.

ALCOHOL

In therapeutics the generic term alcohol refers specifically to *ethyl alcohol* or *ethanol*. Methyl and other alcohols are of only toxicologic importance (p. 756).

The U.S.P. recognizes *grain alcohol* which contains not less than 93.5 per cent of ethanol by weight *dehydrated* or *absolute alcohol* containing not less than 99 per cent and *diluted alcohol* whose strength is not more than 42 per cent.

Alcoholic Beverages.—The systemic effects of alcohol are usually observed through the drinking of alcoholic beverages. Since antiquity fermented drinks have been employed wherever fruit, grain and vegetables have been abundant.

Alcoholic beverages are classified as beers, wines, spirits, liqueurs and cordials. The *beers* contain as little as 3 per cent and the heavier *ales* and *porters* as much as 15 per cent of alcohol. *Wine* may vary in its alcohol content from 7 to 23 per cent; stronger or *fortified wines* contain added ethyl alcohol since yeast fermentation is inhibited in the presence of an alcoholic content in excess of 14 per cent. *Spirits* or *hard liquors* such as whiskey, brandy, rum and gin have an approximate alcoholic content of 50 per cent; the *liqueurs* and *cordials* vary from 32 to 67 per cent of alcohol.

Alcoholic beverages contain extraneous ingredients many of which are responsible for the variables attributed to the alcohol. The reputation of alcohol as a *diuretic* in the beer drinker results from the tremendous intake of fluids rather than any specific effect of the ethanol on the kidney. The mental deterioration of the absinthe drinker is a result of the presence of *uormuood* and the diuretic effect of gin is derived from the volatile *juniper oil*. Other beverage constituents whose effects are not so clearly demonstrable are fusel oil, volatile oils of unknown composition, higher alcohols, aldehydes and the sugars.

The frequency with which alcoholism is associated with limitation of food intake further complicates the evaluation of clinical manifestations attributed to drug effect, so called *alcoholic neuritis* and certain forms of *alcoholic encephalopathy* are more likely caused by vitamin deficiency (p. 622).

Absorption, Distribution, Oxidation and Excretion.—Alcohol is readily absorbed through mucous surfaces. It is demonstrable in the blood stream within a very few moments following ingestion. It is then rapidly distributed to every part of the organism.

The great facility of absorption exceeds the powers of distribution. Blood concentration mounts for at least 60 to 90 minutes. Clinically the effects are observed to steal up on the drinker shortly after he has boasted of his bibulous capacity.

TABLE 21?—THE PHARMACOLOGY AND THERAPEUTICS OF ALCOHOL

| | |
|-------------------------------------|---|
| Reflex Action ¹ | Tingling in mouth increased secretion of mucus saliva and gastric juice local vasodilatation transitory rise in blood pressure pulse and respiratory rates |
| Circulation ¹ | Transitory reflex stimulation and vasodilatation |
| Digestion ¹ | Cocktail or wine before meals increases appetite and liqueurs after meals decrease emptying time copious drinks such as beer diminish food intake and gastritis of chronic alcoholism impairs digestion |
| Central Nervous System ² | General depression pseudostimulation due to "inhibition of inhibitions" later drowsiness stupor and coma. |
| Higher Centers ² | Depression of mental associations concentration idea- tion judgment, reasoning memory and registration of sensory impulses |
| Hypnosis ³ | "Night cap" of ale or whiskey often effectual as a hyp- notic |
| Analgesia ³ | "Hard liquor" often effectual as analgesic in dysmenor- rhea angina pectoris intermittent claudication fatigue headache tabes and insuperable malguancy |
| Metabolism ⁴ | Caloric action useful in fevers for emergency ra- tions |
| Reproductive System ⁵ | Alcohol stimulates libido but depresses coital activity in the timid may be required to inhibit sexual in- hibitions |
| Immunity Mechanism | Not an antidote for snake bite alcoholics resist infec- tion poorly |
| Miscellaneous ⁶ | Does not relieve fatigue or shock increases errors and poisonings in industry obscures injuries in accidents may contribute to medico-legal culpability |

¹ The reflex actions of alcohol have given it a reputation as a *stimulant* and *calcu-
latory stimulant*. The former has some basis in fact but the latter has none.

² The initial depression is manifested on the higher centers which act as monitors to
restrain homo sapiens from behaving in the manner of his boreal ancestor. The depression
of these higher centers where the Freudian Ego (p. 1337) dwells results in a liberation of
the baser Id. There is an inhibition of inhibitions and a resultant pseudostimulation.

"With the depression of the Ego all dreams come true." The inebriate fancies himself
variously as a Don Juan, a giant intellect, a libertine, a child of misfortune, a great humorist
or a tragic figure. The cares, responsibilities and problems of the rest of the world become
unimportant. Restrictions due to legal and moral bans are temporarily lifted; the repressed
libidinal drives into the driver's seat, releases the brakes and steps on the accelerator.

Unfortunately the satanic triumph is short-lived, particularly if the unrepressed Id con-
tinues its patronage of Bacchus. Sooner rather than later the Id and the rest of the cerebral
functions, the cord and the medullary centers fall under the depressant activity of alcohol.
The pseudostimulation gives way to blowsiness, iteration, torpor, stupor and then coma.

³ There is some consolation in the observation that the depressant action of alcohol on
the central nervous system finds therapeutic uses as a *hypnotic* and *analgesic*. Although
the barbiturates are more effectual and less harmful, alcoholic beverages (particularly ale
and porter or the farmhouse "nightcap") often effect a peaceful and restful sleep. While the
practitioner should not sanction the habitual use of the "nightcap," its occasional employ-
ment is often most salutary.

Particularly in protracted illnesses in the aged alcohol increases caloric intake by
stimulating appetite and restoring a sense of well-being.

⁴ The popular conception of alcohol as an aphrodisiac is erroneous in the normal person.
It increases boldness and libidinous thoughts but interferes with the technique of intercourse.
(pp. 2401-2402)

⁵ Most states have heavy penalties for traffic accidents in which alcoholism may be a
contributing factor. Often the drunk is forced on an innocent person after the accident, by
a well-meaning onlooker.

When alcohol is taken in moderate amount it is almost entirely oxidized in the body. With larger amounts oxidation lags and alcohol is excreted by the kidneys and through the lungs. However the odor to the breath of the alcoholic is due very much less to ethyl alcohol than to other constituents in the beverage.

Medicolegal Culpability and Blood Concentration—Because of the medicolegal importance of alcoholism attempts have been made to define the culpability of the drug by objective methods particularly by determining the blood concentration. While there are important individual variations in the response to alcohol there is general agreement that 0.05 per cent or less in the blood is an inconsequential amount. Evidences of a detrimental influence may be assumed to be present when the concentration exceeds 0.15 per cent. In the intermediary zone between 0.05 and 0.15 per cent the extent of the influence of ingested alcohol must be determined by physical examination.

Tolerance and Habituation—The discussion of tolerance and habituation to alcohol is complicated by the psychological aspects of the problem. Like a political discussion this question rarely receives objective debate but is characterized by heated expressions of the prejudices of the staunch defenders and the embittered critics. The physician who attempts to stand between the red and the blue noses finds himself flagellated by both contestants. Conscious of the risk of offending the blue noses it is my belief that significant changes in tolerance do not occur and habituation is not significantly experienced in the normal individual who takes an occasional drink.

Since time immemorial peoples, nations and religious sects have practiced wine drinking with meals and as parts of rites and ceremonies. Despite this the wine drinking peoples do not contribute more than their share of drunkards. The daily intake of alcohol in those of us who can take it or leave it does not significantly vary.

Indubitably the ire of the red noses will be equally aroused by the opinion that the production of increased tolerance to alcohol and of habituation is a manifestation of a psychogenic derangement occurring in constitutionally inferior individuals or those who suffer from profound psychoses.

Toxicology—Alcoholism whether acute or chronic occurs from the abuse of the beverages usually taken without medical supervision or consent.

Acute Alcoholism—Acute alcoholism is a formidable problem particularly referable to traffic accidents and crime. The community must protect itself by penalizing a drunken driver or assailant. The individual protects himself morally and medicolegally by avoiding the ingestion of any alcoholic beverage while operating a motor vehicle.

The *clinical manifestations* of acute alcoholism are unfortunately too familiar. They have been most clearly tabulated by Dr. Walter Miles in the accompanying chart (Table 218).

The *symptomatology* of acute alcoholism does not always run true to pattern. It is determined partially by the personality and character traits of the drinker. In each social group there will be recognized the amiable and the belligerent drunk, the euphoric and the de-

The treatment of acute alcoholism is one of the many nuisances of clinical medicine. Commonly the alcohol intake is automatically checked by the manifestations of toxicology. Gastric intolerance leads to therapeutic vomiting. Somnolence puts an end to the libations and the celebration.

On occasions the physician is summoned to treat an acute alcoholic. These visitations ordinarily occur at some ungodly hour of the night. Under these circumstances it is justifiable to practice sufficient sadism to deter the celebrant from future peccadillos and what is more important

TABLE 218.—CLINICAL MANIFESTATIONS OF ACUTE ALCOHOLISM

| Alcohol in the Blood MG PER CC | Subjective States and Observable Changes in Behavior Under Conditions of Heavy Social Drinking |
|-----------------------------------|---|
| 0.1 | Clearing of the head. Freer breathing through nasal passages. Mild tingling of the mucous membrane of the mouth and throat. |
| 0.2 | Slight fullness and mild throbbing at back of head. Tendency of dizziness. Sense of warmth and general physical well-being. Small bodily aches and fatigue relieved. Not fretful about the weather nor worried concerning personal appearance. Quite willing to talk with associates. Feeling tone of pleasantness. |
| 0.3 | Mild euphoria. Everything is all right, very glad I came. We will always be friends. Sure I will loan you some money. It isn't time to go home yet. No sense of worry. Feeling of playing a very superior game. Time passes quickly. |
| 0.4 | Lots of energy for the things he wants to do. Talks much and rather loudly. Hands tremble slightly. Reaching and other movements a bit clumsy. Laughs loudly at minor jokes unembarrassed by mishaps. You don't think I'm drunk do you? Why I haven't taken anything yet. Makes glib or flippanant remarks. Memories appear rich and vivid. |
| 0.5 | Sitting on top of the world. A free human being. Normal inhibitions practically cut off. Takes personal and social liberties of all sorts as impulse prompts. Is long-winded and enlarges on his past exploits. Can tick anybody in the country but has observable difficulty in lighting a match. Marked flouting of self-criticism. |
| 0.7 | Feeling of remoteness. Odd sensations on rubbing the hands together or on touching the face. Rapid strong pulse and breathing. Amused at his own clumsiness or rather at what he takes to be the perversity of things about him. Asks others to do things for him. Lapses into raving. |
| 1.0 | Stagger very perceptibly. Talks to himself. Has difficulty in finding and putting on his overcoat. Fumbles long with the keys in unlocking and starting his car. Feels drowsy. Sings loudly. Complains that others don't keep on their side of the road. |
| 2.0 | Needs help to walk or to undress. Fairly angered. Shouts, groans and weeps by turns. Is nauseated and has poor control of urination. Cannot recall with whom he spent the evening. |
| 3.0 | In a stuporous condition. Very heavy breathing. Sleeping and vomiting by turns. No comprehension of language. Strikes wildly at the person who tries to aid him. |
| 4.0 | Deep anaesthesia may be fatal. |

After D. Walter Miles. From "Alcohol: Its Effects on Man." New York: D. Appleton Century, 1936.

to protect the physician from the necessity of making future calls of a similar nature.

To that end if the pharyngeal reflex is retained vomiting should be induced by the administration of emetics (p. 1757). If these fail or the patient is uncooperative nothing is more satisfactory to the victimized practitioner and more unpleasant for his recalcitrant patient than the subcutaneous injection of apomorphine 0 mg ($\frac{1}{10}$ grain). Following the emesis even the most belligerent become mollified and soon fall into a deep sleep.

If the patient is agitated an attempt should be made to place him in an improvised continuous warm bath. The oral, rectal or necessary intravenous administration of *paraldehyde* (p 3844) is recommended highly.

Comatose patients may be permitted to sleep it off provided that there are no evidences of systemic difficulties. Recovery from coma is accelerated by the intravenous injection of 50 cc of 50 per cent *dextrose* with 15 units of *insulin*.

As soon as possible *large quantities of fluid* are administered since a negative water balance results from profuse sweating and repeated vomiting. Popular practice to the contrary *purging is to be avoided*. It adds to the dehydration, increases the tendency to acidosis and augments the 'hangover'.

Serious medical situations occasionally arise in association with acute alcoholism. These include the development of an *acute gastritis* and the complications that arise in the treatment of an acute alcoholism in a *diabetic*.

The presence of *gastritis* may be predicated if vomiting continues or lasts beyond twelve hours following the last drink. On occasions the amount of gastritis may be so severe as to cause severe abdominal pain and the manifestations of an *acute abdominal catastrophe* (p 1748), including rigidity of the wall, fever and leukocytosis. Under these circumstances this condition closely simulates the penetration and perforation of a peptic ulcer. Since both conditions may be associated and the findings are closely similar the differentiation may require institutionalization for laboratory investigation. A scout film of the abdomen is taken in the recumbent and erect positions. With perforation there may be free air under the leaf of the diaphragm. An abdominal puncture (p 1920) may reveal foreign material in a perforation. At times though these examinations fail to reveal positive findings the persistence of symptoms warrants a diagnostic laparotomy. Despite the risk of operation in the presence of alcoholism it is wiser to do an unnecessary procedure than to hazard a fulminating peritonitis from delay in the repair of a perforation.

Clinical difficulties arise when acute alcoholism complicates chronic disease particularly *diabetes mellitus*. When this occurs the patient should be given a continuous intravenous drip containing glucose solution covered by insulin in the proportion of 1 unit per 15 to 2 gm of sugar. The continuance of the drip is guided by the same principles as those used in the treatment of acidosis (p 721).

Following acute alcoholism the familiar symptoms of the hangover occur. These include nausea, distaste for food, irritability and usually a splitting headache.

The popular *prophylactics and remedies* for the hangover include the liberal ingestion of fluids such as cold tomato juice, the use of thiamin chloride by mouth or intravenously and dosage with the popular effervescent analgesic antipyretics. The important physiological principles are the relief of dehydration by copious water drinking, the restoration of the electrolyte balance by a generous intake of alkali and protection of the insulted mucous membranes by frequent feedings of high calory starch and carbohydrate foods.

Chronic Alcoholism—While an occasional experience with acute alcoholism is probably the right and privilege of every adult the problem of chronic alcoholism is a far different matter. Chronic alcoholism is in my opinion not so much the concern of the pharmacologist or physician as it is the province of the psychiatrist.

Fundamentally *the chronic alcoholic is not a normal person*. He is a constitutional inferior or a victim of his own psychopathic personality. The drug poisoning is but one symptom of the underlying complaint. Alcohol cures directed at the pharmacological problem are poorly devised and practically useless.

The extent of the problem of chronic alcoholism cannot be overestimated. Each practitioner knows that in an inordinate percentage of his families there is at least one sot. Oftentimes the family drunk beclouds and ruins the lives of the immediate household.

THE VARIETIES OF CHRONIC ALCOHOLISM—Chronic alcoholism may manifest itself as a *constant state of inebriety with deterioration and progressive impairment of all functions*. The psychosis may take on the characteristics of that described by Korsakoff in which the deteriorated patient has only a memory of the remote past. It may be a specific type of *encephalopathy* (p. 1384) probably due to nicotinic acid deficiency (p. 620) manifested by clouding of consciousness, cogwheel rigidity and uncontrollable grasping and sucking.

The chronic psychoses are usually punctuated by episodes of *acute alcoholic hallucinosis*. These involve visual mirages (pink elephants) associated with generalized tremulousness (*delirium tremens*). There may be *mania*, *paranoia* or *coma* (*wet brain*).

In *dipsomania* the drinking bouts alternate with periods of complete sobriety and even evangelism.

The psychiatric manifestations of chronic alcoholism are usually associated with organic disorders such as *peripheral neuritis*, *cirrhosis of the liver*, a *generalized cachexia or malnutrition* and the *beriberi heart*. These are most likely manifestations of an *avitaminosis* rather than a consequence of the alcoholism per se. They occur much more commonly in the indigent alcoholic whose money is expended entirely on his booze. They are less often experienced in the affluent drunk who has additional funds with which to pay for edibles.

TREATMENT OF CHRONIC ALCOHOLISM—The treatment of chronic alcoholism is as important as it is unsatisfactory. The most important principle is the necessity for institutionalization. It is difficult to get the patient or his family to agree to this procedure. It is quite impossible for the practitioner to cope with the chronic alcoholic. He has neither the time nor the extra energy to stand guard over his patient. He cannot continue his attentions over a sufficient length of time without neglecting the rest of his practice. Sooner or later he becomes discouraged, disgusted and thoroughly disheartened.

The ordeal that the physician experiences is trivial compared to the suffering endured by the remaining members of the household. The parents and marital partner of the drunkard sooner or later succumb to the unequal struggle. Children become demoralized and tremendous sacrifices are made by the family without avail. *For the protection of the patient*

his family the community and the practitioner the alcoholic should be institutionalized for a period of at least six months and preferably one to two years. Institutions which manage alcoholics are unfortunately mostly of an inferior caliber. It is the duty of the clinician to investigate the psychiatric hospitals in the vicinity and find one to which the patient can be conscientiously referred.

The immediate therapeutic problem in the management of the chronic alcoholic whether within or without the institution is concerned with the treatment of the presenting symptoms withdrawal of alcohol in any form and mental and physical rehabilitation.

The comatose drunk presents a less trying problem than one in an agitated state. During the period of coma a continuous intravenous drip is instituted. The infusate consists of 5 per cent glucose in saline solution. Insulin is added in the proportion of 1 unit per 15 to 2 gm of sugar. Thiamin chloride crystals are included in 100 mg doses.

On awakening the comatose alcoholic usually becomes violent and then is managed in the same manner as the agitated drunk that is he is placed as soon as possible in a continuous warm bath (p 3133). While this can occasionally be accomplished by persuasion and a little force it is usually necessary to use sedatives. Paraldehyde is the preparation of choice. It may be administered orally in beer rectally in a starch retention enema or intravenously when the patient refuses to cooperate or is unable to retain the dose at either end of his digestive tract. Since the alcoholic develops a gross tolerance for paraldehyde it is often necessary to give seemingly colossal doses such as 30 to 60 cc by mouth 60 to 120 cc by rectum and 2 to 10 cc intravenously.

The substitution of chloral and the barbiturates may lead to clinical difficulties since these drugs produce untoward side effects adding to the presenting problem. The use of morphine is greatly to be deprecated. Attempts to stimulate the cerebrum with caffeine and amphetamine sulfate are more likely to add to the dilemma than to effect any amelioration of the situation. Amphetamine sulfate has been used successfully in alcohol addiction by using it as a benign substitute addiction.

Drainage of spinal fluid through lumbar puncture is recommended by many clinicians particularly during the phases of wet brain. Other experienced psychiatrists are opposed to lumbar puncture except for diagnostic reasons. The latter opinion corresponds with my own views.

Sooner or later the recovering patient begins his demand for a drink. Opinions differ as to procedure. Many clinicians believe that complete withdrawal increases agitation and hazards the institution of a period of delirium tremens. It is their practice to give decreasing amounts of diluted alcohol over the period of the first few days of treatment. Others whose opinions I favor practice complete withdrawal of alcohol. A firm statement of this purpose puts an end to the pleadings and cajoling of the patient to whom paraldehyde may be administered as soon as agitation commences.

The sanitariums which specialize in alcohol cures place great reliance on specific drugs which are usually belladonna or one of its derivatives. The administration of the specific is usually accompanied by excessive purging. This type of cure has no excuse for its existence either in theory or practice. The belladonna poisoning adds to the confusion. Purging tends further to dehydrate the patient.

abohic difficulties. Emetine is used in conjunction with alcohol to set up a conditioned reflex of vomiting in order to create an aversion to liquor. This method has had favorable reports but is still under trial.

As soon as the acute disturbances are controlled the patient is put on a *high-calory high carbohydrate soft diet* containing an abundance of starches, carbohydrates and the protein foods. All of the *synthetic vitamins* are added. Wherever possible these are given parenterally since absorption from the digestive tract may be irregular at best.

The patient is urged to drink as much fluid as is possible. Citrus and carbonated drinks are favored because of their vitamin content and their alkalinizing effects.

Physiotherapy is continued. The continuous bath is replaced by the *Scotch douche*, *massage* and *graded exercises*. Later the patient is encouraged to engage in *occupational therapy*. The physical rehabilitation is accompanied by *expert psychotherapy*.

The complexity of the treatment of chronic alcoholism requires institutional care. In the home improvisations are bound to fail. Moralizing, preaching, the taking of vows and temperance oaths, appeals to decency, arguments containing profoundly good sense, may all as well be written in the sand. It is one matter to put the drunk on the wagon. It is a far more difficult problem to persuade him to face a world of reality rather than one that is peopled by the inhabitants created in his wet brain. Occasionally abstinence groups such as the *Alcoholic Anonymous*, consisting of reformed drunks, are more helpful to the patient than efforts by those who have not had the personal experience.

If by great good fortune a successful outcome has been accomplished, arrangement should be made whereby the individual returns to the institution for a week or two weeks every few months. This is an important prophylactic in preventing the alcoholic from slipping from grace.

OPIUM AND THE OPIATES

Opium is nature's great gift to suffering humanity. The rapid relief of pain that follows the administration of opium and the opiates is a therapeutic achievement of incalculable value.

Opium, the crude drug, is derived from the dried milk juice of the unripe seed capsule of the *white poppy* (*Papaver somniferum*). The white poppy is cultivated chiefly in Turkey, India, Egypt and Peru. More than twenty alkaloids, constituting 25 per cent of the weight of the crude drug, have been isolated from opium. The most important of these are *morphine*, which is present in greatest amount (10 per cent of the total alkaloids), *codeine* (0.5 per cent) and *papaverine* (1 per cent). The synthetically prepared opiates include *dionin*, *heroin*, *codeine*, *apomorphine* and *dilaudid*.

Opium Alkaloids.—The opium alkaloids fall into two groups, distinct chemically and pharmacologically. The *phenanthrene compounds* (*morphine*, *codeine*) act chiefly on the central nervous system and stimulate smooth muscle. The *isoquinoline group* (*papaverine*) have a negligible action on the nervous system and depress smooth muscle.

Preparations and Doses.—The principal preparations and dosages of the opiates are shown in Table 220.

TABLE 219—THE PRINCIPAL ALKALOIDS OF OPIUM

 C_0 of Total Alkaloids

Phenanthrene Derivatives

| | |
|----------|------|
| Morphine | 10.0 |
| Codeine | 0.8 |
| Thebaine | 0.2 |

Isoquinoline Derivatives

| | |
|------------|-----|
| Papaverine | 1.0 |
| Narcotine | 0.0 |
| Narcine | 0.3 |

PHENANTHRENE



ISOQUINOLINE



TABLE 220—PRINCIPAL PREPARATIONS OF OPIUM AND ITS DERIVATIVES

| Preparation | Active Ingredient | Remarks |
|---|---|---|
| Purified Opium USP | 0.06 gm (1 gr) as pill or suppository | Eq. lent to 5 mg (1/2 gr) of morphine. |
| Powder of Precipitated Opium USP (Dose: Powder) | 0.5 to 0.6 gm. by mouth (5-10 gr) | Contains 10% each of peacodone, Eq. lent to 5-6 mg (1/4 gr) morphine. |
| Tincture of Deodorized Opium USP (Laudanum) | 0.5 to 2.0 cc by mouth | A 10% alcohol solution, Eq. lent to 1 morphine. |
| Concentrated Tincture of Opium USP (Pregnac) | 4 cc by mouth | Contains 0.4% opium, 0.04% morphine, also 1% anise, benzoic acid, camphor. 4 cc. corresponds to 1.6 mg (1/4 gr) morphine. |
| Compound Mixture of Opium and Glycyllate USP | 4 cc by mouth | Contains 10% peacodone, 1% morphine, 1% dextro-pseudoephedrine. |
| Morphine Sulfate USP Morphine Hydrochloride NF | 8 to 20 mg (1/4 to 1/2 gr) by mouth 8-15 mg (1/4 to 1/2 gr) subcutaneously 2.5-15 mg (1/4 to 1/2 gr) transdermally (rely on assay) if ill 1 liter of 10 mos of gestation dose is | Contra-indicated in conditions where case of peacodone has been observed. |
| Mg. l. Solution | 7 m m (eq. 1 to 15 mg) gr n f m rpl e | Contains morphine sulfate 33 gm, analgic acid 0.1 gm, 1 liter to 100 cc. |
| Dextrolym rpl e Hydrochloride (Hydrochloride) | 2.5 to 8 mg (1/4 to 1/2 gr) | Important in USA because of dextrolym. |
| Dextrolym rpl e Hydrochloride (Dextrolym NF) | 1.5 mg (1/4 gr) by mouth, subcutaneously | Analogous to 10 times greater than morphine. Dextrolym is 10 times more potent than morphine. Dextrolym is 10 times more potent than morphine. Dextrolym is 10 times more potent than morphine. |
| Pitip (Pitip m Hydrochloride) | 20 mg (1/2 gr) by mouth, subcutaneously | A purified mixture of opium alkaloids. Contains 80% opium, 1% dextrolym, 1% morphine, 1% codeine. |
| Dextrolym rpl e Hydrochloride USP | 10 to 20 mg sol in p crystals | Used in ophthalmology. |
| Cod. Sulfate USP Cod. e Phosph. USP | 8-60 mg (1/4 to 1 gr) by mouth, subcutaneously | Specific effect on cough, but less than one of the narcotic potencies of morphine. Rarely used as expectorant if because of greater solubility. |
| Papaverine Hydrochloride NF | 30-80 mg (1/2 to 1 1/2 gr) by mouth, subcutaneously, 10-60 mg (1/4 to 1 gr) intravenously | Relates to spasms of vessels, dilates blood vessels, dilates pupil. |
| Apomorphine Hydrochloride USP | 3-5 mg (to 1/4 gr) by mouth, subcutaneously, 1 mg (1/4 gr) intravenously | Contra-indicated in some cases of acute pulmonary edema. Sol. in liquid of the used if it has been dried. |

NOTES ON THE OPIUM PREPARATIONS

Powdered opium is best employed as a suppository for oral use *paregonic* is preferable *Dover's powder* has little to recommend it To produce diaphoresis the prescription of a nauseant (ipecac) is ill advised Recently *Dover's powder* has been supplanted by the proprietary *Copaxin* which contains codeine and papaverine *Propose 1* as a cold specific *Copaxin* like *Dover's powder* also contains ipecac
The deodorized tincture of opium is best replaced in the household medicine cabinet by the weaker preparation of *paregonic* for while opium poisoning may be caused by the ingestion of a few ounces of laudanum a similar mistake with *paregonic* need not be feared

Brown mixture is a needlessly complicated preparation The use of niter and tartar emetic as diaphoretics and expectorants is objectionable The ammonium salts are equally efficacious and completely free from untoward side effects A simpler and more effective opium prescription for the relief of cough contains codeine phosphate as the active alkaloid elixir of terpene hydrate or syrup of wild cherry as the vehicle and *Liquor Ammon. Anisatus* as a liquefying expectorant

Tablets of *morphine sulfate* should be carried in the pure state If atropine or scopolamine is to be administered simultaneously it too should be available in pure form It is rarely necessary or advisable to repeat the doses of atropine or scopolamine while morphine may be indicated several times When the alkaloids are present in the same tablet it is not possible to administer one without the other

Magendie's solution is used in hospital practice A sterile solution of morphine sulfate marketed in one-ounce bottles with a rubber stopper may be purchased for office use While the cost of each dose of morphin is necessarily increased precious seconds and minutes are saved in administering the dose Morphine solutions are given intravenously in situations of grave emergency

Heroin (diacetylmorphine) is no longer available in the United States because of its high favor in drug addiction From a medical point of view this is unfortunate since this preparation was very effective for the relief of cough

Dilaudid (dihydromorphine) seems to have many practical advantages over morphine It is less likely to produce nausea vomiting and addiction It is more analgesic than morphine and has less of a tendency to cause drowsiness

Pantopon behaves like morphine It is a mixture of purified opium alkaloids Its increased cost is not warranted

The use of *dionin* (ethylmorphine) is limited to local application in the conjunctival sac for the production of vasodilatation

Codeine (methylmorphine) is used if possible before resorting to morphine *dilaudid* or *pantapon* In painful conditions it is futile to give less than 30 mg ($\frac{1}{4}$ gr) to the dose and 60 mg (1 grain) is preferable The combination of codeine and acetylsalicylic acid is so effective as to suggest a synergism Doses of codeine in excess of 120 mg (2 grains) may cause cortical excitation and stimulation of the cord

Lapomrine like codeine is underestimated clinically because of the tendency to administer insufficient doses When given intravenously in dosages of 30 to 120 mg it may be efficacious in relieving vasospastic conditions (p. 791)

The use of *apomorphine* is limited to the production of emesis in conscious patients

PRESCRIPTIONS CONTAINING OPIUM AND ITS DERIVATIVES

For Acute Upper Respiratory Infections (Common Cold)

| | |
|------------------------------------|------|
| Codeine Sulfate | 0.25 |
| Papaverine Hydrochloride | 0.25 |
| Make 15 capsules | |
| Take one every 4 hours for 4 doses | |

| | |
|------------------------------------|------------|
| Codeine Sulfate | 0.25 |
| Papaverine Hydrochloride | 0.25 |
| Sodium Salicylate | 200 |
| Syrup of Wild Cherry | qs ad 2400 |
| Take a tablespoonful every 4 hours | |

For Cough

| | |
|----------------------------------|--------------|
| Codeine Phosphate | 0.25 |
| Elixir of Terpene Hydrate | q.s. ad 60.0 |
| Take 1 teaspoonful every 3 hours | |

OR

| | |
|--|--------------|
| Codeine Phosphate | 0.25 |
| Liquor Ammonii Anisatus | 20.0 |
| Syrup of Wild Cherry | q.s. ad 60.0 |
| Take a teaspoonful in $\frac{1}{4}$ glass of water every 4 hours | |

For Pain Accompanied by Muscle Spasm (Renal Colic)

| | |
|------------------------|-------|
| Morphine Sulfate | 0.25 |
| Atropine Sulfate | 0.015 |
| Divide—make 15 tablets | |

For Pain and Vomiting

| | |
|------------------------------|------|
| Codeine Phosphate | 1.0 |
| Acetylsalicylic Acid | 10.0 |
| Divide—make 15 suppositories | |

For Pain Associated with Motor Excitement

| | |
|--------------------------|-------|
| Morphine Sulfate | 0.25 |
| Scopolamine Hydrobromide | 0.005 |
| Make 15 tablets | |

For Diarrhea

| | |
|---|--------------|
| Tincture of Opium | 20.0 |
| Syrup of Chloroform | q.s. ad 60.0 |
| Take a teaspoonful as required dilute with water if necessary | |

OR

| | |
|----------------------------------|--------------|
| Camphorated Tincture of Opium | 10.0 |
| Chalk Mixture | q.s. ad 60.0 |
| Take a teaspoonful every 3 hours | |

Absorption Fate and Excretion—Opiates enter the body readily through the gastro intestinal tract or *parenterally*

Morphine is largely destroyed and detoxified in the body Only 10 per cent is excreted in the urine *Papaverine* like morphine is probably destroyed in the body though its fate is not definitely known *Codeine* is chiefly disposed of by the kidney 80 per cent being eliminated in the urine

The effects of opiates are prolonged to the point of toxicity in *hepato renal insufficiencies* The reduction in excretion and the rate of detoxification result in cumulation when repeated doses are given *Codeine is especially to be feared in kidney disease Morphine is more toxic in liver disturbances*

TABLE 221.—THE PHARMACOLOGY AND THERAPEUTICS OF OPIUM AND THE OPIATES

Pain Center

Analgesia is produced centrally by elevation of the pain threshold and by alteration of the quality of the painful stimulus

Analgesia is the prime indication for the use of the opiates Preferably the drug is used to ease acute severe pain that is accompanied by or productive of shock and anxiety Relief of pain may be an undesirable virtue in the management of undiagnosed abdominal catastrophes In these situations the opiate clouds the clinical picture and urgent and necessary surgery may be delayed to the detriment of the patient In vir-

TABLE 291—THE PHARMACOLOGY AND THERAPEUTICS OF OPIUM AND THE OPIATES (Continued)

tually all other clinical conditions the relief of pain is an unmitigated blessing. The analgesia affords subjective relief and it may prevent physiological derangements such as resultant shock and spare the circulatory system by removing the load imposed by the attendant agony and anxiety. The experience of war has demonstrated the lessened incidence of shock when soldiers are provided with the means of making injections of morphine immediately upon receipt of an injury.

It is almost an act of ingratitude to stress the point that opiates do not relieve the fundamental cause of pain. In point of fact, the smooth muscle stimulant action of morphine tends to accentuate the deranged mechanisms in colic and angospastic conditions. Because of this atropine is injected simultaneously to assist the relaxation of smooth muscle or papaverine or demerol is substituted.

Subcutaneous injection has an effect in ten minutes but does not reach the peak for thirty to sixty minutes. Intravenous administration gives immediate relief with a peak in twenty to thirty minutes. The latter may permit much smaller effectual dosage, more rapid and complete benefits and less hazard of cumulative poisoning, particularly relative to the respiratory center.

The analgesic effect is potentiated by scopolamine (p. 386) producing twilight sleep. This combination is extensively used for pre-anesthesia, basal anesthesia and obstetric anesthesia.

Morphine and the opiates are not hypnotic. On the contrary they may create a state of wakefulness that is associated with a "peace that passes all understanding." The reputation of morphine and the opiates as hypnotics is due to the sleep that comes to patients who have been wracked with pain or distress for many hours or days. In these instances the sleep is not a direct action of the drug but follows relief of symptoms.

Respiratory Center

Respiratory depression is due to lessened sensitivity of the center to anoxia and hypercapnia.

The use of opiates in cardiorespiratory disease is a two-edged sword. In the presence of rapid shallow breathing, opiate has great therapeutic value. Prior to its use the patient may be dyspneic, restless, sleepless and anxious. In part the distress results from scapnia and anoxemia, since hyperventilation leads to increased exhalation of carbon dioxide and inadequate oxygenation. After the use of the drug the respiratory rate is slowed and breathing is deeper. The blood is more effectively laden with oxygen and an adequate concentration of carbon dioxide is maintained. These changes are reflected by a lessening of the dyspnea, diminution of anxiety, a dissipation of the sense of suffocation and a gratifying improvement in general circulatory efficiency. The patient may indulge in the luxury of a restful sleep and awaken greatly refreshed.

In contrast to the beneficial effects of opiate in hyperventilation are the toxic manifestations referable to depression of the respiratory center. With excessive dosage, impairment of excretion or unusual sensitivity of the medulla, the patient may develop respiratory irregularities, particularly Cheyne-Stokes respirations and progressive cardiorespiratory failure.

Cough Center

Depression is a central effect and secretions may collect in respiratory passages.

Depression of the cough reflex is particularly undesirable in atelectasis (p. 2052) and bronchial asthma (p. 2101). The secretions become more viscous and may form obstructive casts.

Vomiting Center

Stimulation with frequent emesis occurs constantly and within a few minutes from the administration of 6 mg. (two grains) of apomorphine. This action is useful in the treatment of poisonings and alcoholism. Morphine emesis may be delayed many hours and occurs in 1 out of every 3 or 4 patients. Delayed morphine vomiting must not be confused with emesis due to underlying disease or prior operative procedure.

For Cough

| | |
|----------------------------------|-----------|
| Codeine Phosphate | 0.25 |
| Elixir of Terpene Hydrate | qs ad 600 |
| Take 1 teaspoonful every 3 hours | |

OR

| | |
|--|-----------|
| Codeine Phosphate | 0.25 |
| Liquor Ammonii Anisatus | 200 |
| Syrup of Wild Cherry | qs ad 600 |
| Take a teaspoonful in $\frac{1}{4}$ glass of water every 4 hours | |

For Pain Accompanied by Muscle Spasm (Renal Colic)

| | |
|------------------------|-------|
| Morphine Sulfate | 0.25 |
| Atropine Sulfate | 0.015 |
| Divide—make 15 tablets | |

For Pain and Vomiting

| | |
|------------------------------|-----|
| Codeine Phosphate | 10 |
| Acetylsalicylic Acid | 100 |
| Divide—make 15 suppositories | |

For Pain Associated with Motor Excitement

| | |
|--------------------------|-------|
| Morphine Sulfate | 0.25 |
| Scopolamine Hydrobromide | 0.003 |
| Make 15 tablets | |

For Diarrhea

| | |
|---|-----------|
| Tincture of Opium | 200 |
| Syrup of Chloroform | qs ad 600 |
| Take a teaspoonful as required dilute with water if necessary | |

OR

| | |
|----------------------------------|-----------|
| Camphorated Tincture of Opium | 100 |
| Chalk Mixture | qs ad 600 |
| Take a teaspoonful every 3 hours | |

Absorption Fate and Excretion—Opiates enter the body readily through the gastro intestinal tract or *parenterally*

Morphine is largely destroyed and detoxified in the body. Only 10 per cent is excreted in the urine. *Papaverine* like morphine is probably destroyed in the body though its fate is not definitely known. *Codeine* is chiefly disposed of by the kidney, 80 per cent being eliminated in the urine.

The effects of opiates are prolonged to the point of toxicity in *hepato renal insufficiencies*. The reduction in excretion and the rate of detoxification result in cumulation when repeated doses are given. *Codeine* is especially to be feared in kidney disease. *Morphine* is more toxic in liver disturbances.

TABLE 221 —THE PHARMACOLOGY AND THERAPEUTICS OF OPIUM AND THE OPIATES

Pain Center

Analgesia is produced centrally by elevation of the pain threshold and by alteration of the quality of the painful stimulus.

Analgesia is the prime indication for the use of the opiates. Preferably the drug is used to ease acute severe pain that is accompanied by or productive of shock and anxiety. Relief of pain may be an undesirable virtue in the management of undiagnosed abdominal catastrophes. In these situations the opiate clouds the clinical picture and urgent and necessary surgery may be delayed to the detriment of the patient. In vir-

Choice of Preparation—There is an increasing tendency to withhold the use of *morphine* if possible until other analgesic measures have been tried. Nowhere is this better illustrated than in modern surgery where most conscientious practitioners attempt to curtail the use of the drug before and after operation. The undesirable features of morphine administration include its tendency to encourage gastric dilatation, intestinal ileus and urinary retention. It is a frequent cause for postoperative vomiting and the inevitable itching may be extremely annoying.

In protracted illness the liberal use of morphine often given with humanitarian purpose may add appreciably to the clinical burden. The patient becomes more demanding for the relief of his pain. Injections have to be given at more frequent intervals and in larger doses the appetite fails, the bowels move poorly, urination is difficult, scratching is maddening and there is general physical and mental deterioration with the danger of addiction. Despite all these deterrents morphine is the practitioner's ace for the relief of acute and violent pain although demerol (p. 3863) looms as a potential rival.

The substitution of *codeine* for morphine is often desirable. The analgesic effect of the drug is much less but so too are the side effects. Codeine is particularly recommended as a cough sedative. *Opium* finds its greatest use in the control of diarrheas. Small doses are sometimes included in cough syrups but this practice is not one to be encouraged. Except for its emetic action *apomorphine* has no place in clinical therapeutics. *Dionin* is used only for its local irritant effect in the eye and *heroin* can no longer be manufactured or sold in the United States.

Our experience with *pantopon* has been thoroughly unsatisfactory. It may be regarded as a weak morphine solution with no fewer untoward side effects. We have had greater success with *dilaudid* which appears to us to be as effective an analgesic with considerably fewer unsatisfactory side-effects and a slight possible tendency toward the development of addiction.

Papaverine—Papaverine, a benzylisoquinoline derivative of opium, is unrelated to morphine and does not cause depression of the central nervous system. To the contrary, large amounts of the drug increase reflex excitability.

The chief action of papaverine is depression of smooth muscle, particularly of the blood vessels, bronchi, gastro-intestinal and biliary tracts and the ureters. This action is more apparent in the presence of spasm. The muscle cells are not paralyzed but are capable of responding to stimuli.

From a therapeutic standpoint papaverine has a limited value. The best results are obtained in patients with acute peripheral vascular occlusion after injections of 60 to 120 mg. of papaverine hydrochloride intravenously. It counteracts the reflex vasospasm incident to the local vascular accident and favors the development of a collateral circulation. Good results have been reported in patients with pulmonary embolism, Raynaud's disease and in coronary occlusion. It may be tried in epinephrine-fast asthmatics and in biliary and renal colic. Its toxicity is low and there is apparently no tolerance or habituation.

Contraindications—The use of morphine and the opiates is contraindicated in a variety of conditions. They are particularly avoided in patients

TABLE 221 —THE PHARMACOLOGY AND THERAPEUTICS OF OPIUM AND THE OPIATES (Continued)

Circulation

Morphine and the opiates have no direct effect on the circulation other than a transitory and unimportant cutaneous vasodilation

Stimulation of vasomotor center causes flushing of face

Central Nervous System

The normal and usual response is *cortical depression* but excitation is encountered as an occasional idiosyncrasy (p 3815)

Pupillo constrictor center is stimulated causing pin point pupils

Spinal cord is stimulated with increase in deep reflexes as delayed effect ("fro action")

May cause restlessness and induce repetition of dose with hazard of initiating habituation (p 3861)

Morphine and the opiates are used sparingly in neurological and psychiatric conditions. In the treatment of patients afflicted with conditions of *hyperreflexia* or a tendency to *convulsions* the stimulant effect of morphine on the cord may be most unfortunate. Morphine is a potentially harmful drug in the treatment of most psychiatric conditions particularly the *psychoses* and *hysteria*. Patients with these disorders are candidates for addiction. Often they feign renal or biliary colic in order to obtain an extra injection of the drug. Morphine and its substitutes are also to be used most judiciously in the treatment of *migraine*. Many addicts who attributed their difficulty to the legitimate use of the drug by a practitioner received their first injections for the relief of headache or a migraine equivalent.

Smooth Muscle

Morphine increases the tonus and contractility of longitudinal muscle and causes spasm of sphincters. It may cause gastric dilatation, intestinal ileus, obstipation and urinary retention. It impedes the passage of a calculus and may increase colic. Except in peritonitis the smooth muscle action of morphine is undesirable.

Papaverine relaxes smooth muscle but intravenous doses of 60 or 120 mg (1 or 2 grains) are required prefer to morphine in colic and angospastic disease (p 91)

Metabolism

Basal metabolic rate and body temperature depressed but blood sugar rises

Skin

Intense pruritus a persistent and annoying side effect

Idiosyncrasy—Morphine idiosyncrasy is encountered as a species and an individual variant. When morphine is given to a *dog* it vomits, curls up in the corner and sleeps until the effects have worn off. A *cat* becomes violently maniacal and may develop convulsions simulating those seen in strychnine poisoning. The *frog* after an initial depression develops delayed hyperirritability and marked stimulation of the cord and the deep reflexes.

Dog, cat and frog actions are exhibited by human beings. In our experience the majority of morphine reactions are dog like except that the incidence of vomiting is not universal. The cat action is not uncommon; many patients reporting relief of pain accompanied by a state of alert and excitement. Those who make careful observations of their patients will frequently encounter frog like responses: the patient reports a late increase of restlessness and irritability and may demand another shot to take the edge off the nerves. If there is not an accompanying degree of significant pain the wary physician recognizes this as a danger signal since it presages addiction. The patient should receive a "shot" but the injection should be of sterile water or a soluble b

ment in the milder degrees of morphine poisoning. The respiratory rate may fall to 16 12 10 8 and even 6 to the minute without other objective signs or subjective discomfort. So long as the patient is at peace and the circulatory mechanism is functioning adequately, antidotal therapy is unwise and gratuitous and may in fact be productive of considerable damage.

It is only when the respiratory depression causes demonstrable changes such as increase in cyanosis, elevation of pulse rate or evidences of circulatory failure that treatment should be instituted.

I am of the belief that *only the simplest measures should be employed in the treatment of morphine poisoning*. The patient is kept warm and artificial respiration maintained by manual efforts or mechanical devices. Oxygen is administered by nasal catheter or the use of a mask. There is no need of giving carbon dioxide since the blood contains an excessive amount.

If the opiate has been taken by mouth gastric lavage is advisable. An intravenous injection of 50 cc. of 50 per cent dextrose stimulates urinary excretion without adding to the circulatory load of the heart.

I am opposed to the measures of treatment popularly advocated. Flagellation of the unfortunate patient in an effort to keep him awake and the administration of toxic amounts of atropine, caffeine, strychnine and the medullary convulsants (coramine, metrazol and the like) add to the difficulties of the situation. If breathing and oxygenation cannot be maintained by artificial respiration and the inhalation of the gas, none of the heroic pharmacological antidotes can be expected to work and their side effects only increase the burden.

The vast majority of patients with morphine poisoning induced through medical means will recover provided that their antidotal treatment is not too excessive. I have never in my own experience encountered a death from morphine given for therapeutic purposes. When excessive doses of morphine (more than 0.25 gm. {4 gr.}) are taken with suicidal intent the prospect of recovery is not good. The chances are not increased by over-enthusiastic poisoning with the alkaloidal stimulants.

Morphine Addiction—Tolerance to opiates and morphine occurs with great facility. Opium and morphine require increasing dosage after regular administration for a few weeks. Codeine and dilaudid exhibit a lesser tendency to toleration but increasing dosage is demanded when either of these drugs is given regularly over the course of several months.

As with alcohol, morphine addiction does not occur in the normal individual who desires to refrain from the use of the drug as soon as the indications disappear. Stable persons have no craving to resume opiate except under repetition of physical anguish. Sufferers from chronic painful conditions of hopeless prognostic implication do become addicted of course but only because life is not otherwise endurable.

In the absence of painful affliction, morphine addiction is a symptom of an underlying psychogenic disorder rather than a disease per se.

The symptoms of morphine addiction are variable and are rarely due to the administration of the drug. The morphinist may appear to be no different from other members of the population and pursue his regular daily activity without arousing the slightest suspicion of his drug addiction.

with *disturbances of liver function* (p 1953) Codeine is used with caution in *renal insufficiency* (p 2276)

The opiates are employed sparingly in *respiratory depression* and conditions associated with *cyanosis*. The addition of the morphine effect on the medullary center may add to its burden. The drug decreases the respiratory rate lessens the chance of adequate oxygenation of the blood and favors the accumulation of carbon dioxide. As a result, respiratory irregularities of the Cheyne Stokes type may be produced. Death may occur from respiratory failure the heart beating for several moments after the terminal apnea.

Opiates are avoided in *abdominal catastrophes* until a definitive diagnosis has been reached.

The use of morphine and the opiates may be hazardous when given in association with *depressant or hepatotoxic drugs*. It is unwise to administer morphine when basal anesthesia is to be effected with avertin. Opiate poisoning is more easily induced in patients receiving *sulfonamides*. The simultaneous use of the two powerful pharmacological preparations requires the most judicious supervision.

Morphine is contraindicated in the treatment of conditions associated with *itching* (p 3170).

Toxicology—The toxic manifestations of the administration of the opiates result from (1) *idiosyncrasy* (2) *acute poisoning* or (3) *addiction*.

Idiosyncrasy—Idiosyncrasy to the opiates occurs with greater frequency than is generally recognized. The production of nausea vomiting and intractable itching has only nuisance value. The gastric disturbances are important to recognize in the management of cardiac and surgical patients. If they are erroneously interpreted they lead to serious blunders in management. In *cardiac patients* the vomiting may be attributed to hepatic engorgement or digitalis poisoning. In the *surgical patient* a local complication is often feared. Errors are more easily made when as often happens there is a latent period of several hours between the injection of the opiate and the production of the emesis.

The *cat reaction* (motor and cerebral excitation resulting from morphine) occurs much more often than is commonly believed. A certain number of patients warn the physician that they have had the experience of going out of their heads from a hypodermic injection. The cat reaction must be recognized lest additional morphine be injected to relieve the very symptoms which the drug has caused.

Acute Morphine Poisoning—The ominous evidences of morphine toxicity are referable to the *respiratory center*. The depression is noted clinically by decreasing respiratory rate and the presence of irregularity in breathing.

The toxic effect on the respiratory center may result from overdosage, accumulation or diminished destruction and excretion in *hepatorenal insufficiency*. The respiratory center seems more apt to be depressed when avertin and sulfonamides are administered simultaneously.

The diminution in respiratory rate need cause no concern so long as the patient's general condition remains excellent. One of the most fascinating clinical observations is the lack of distress or circulatory embarrassment.

drip may be started. From then on the patient is kept quiet by the introduction into the infusion of paraldehyde or soluble barbiturate.

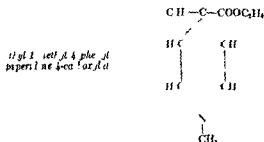
When the acute period has passed the treatment of morphine addiction consists in *physical and mental rehabilitation*. Metabolic deficiencies and avitaminoses result in the morphinist for the same reason that they exist in the alcoholic. The indigent morphinist spends all of his income on his drug and has no money and no appetite for food. It is such a one that exhibits the familiar textbook picture of the addict. The more affluent morphinist gets sufficient food to maintain his metabolic needs and often looks as well fed and normal as any of his colleagues in similar walks of life.

Indigent morphinists occasionally become *main line shooters*. This variety of addict takes the drug intravenously for rapidity of action and economy. Since part of the code of the morphinist seems to be never to sterilize a needle or syringe, epidemics of infectious syphilis and malaria may follow the use of a community syringe.

The rehabilitation program of the addict includes the use of a *high calory diet*, *physiotherapy* and *exercise therapy* to build up the bodily musculature, *occupational therapy* and skillful *psychotherapy* for the restoration of morale.

DEMEROL

An important addition to the group of analgesic drugs is demerol, a synthetic compound which resembles atropine in chemical structure. Because of its marked analgesic effect it has been advocated as a morphine substitute. Demerol is ethyl 1-methyl-4-phenylpiperidine-4-carboxylate and has the following structural formula:



Pharmacology—Demerol possesses the ability to depress the central nervous system resulting in *analgesia* and *sedation* and exhibits a depressant action on smooth muscle.

Central Nervous System Depression—Demerol produces a morphine-like analgesia in man. An oral dose of 100 mg. leads to an elevation of the pain threshold in fifteen minutes which persists for several hours. Visceral pain such as that arising from the peritoneum, pleura or smooth muscle is relieved more effectively than pain arising from skeletal and nervous structures. Comparative studies indicate that 50 mg. intramuscularly are more potent than 22 mg. of codeine. 100 mg. parenterally are equal to 10 mg. of morphine.

Smooth Muscle Action—Demerol has a complex depressant action on smooth muscle which results in a marked spasmolytic effect due to an

The popular concept of the morphine addict is seen only in the deteriorated underprivileged subject. The patient is lean and poorly nourished and there is marked tremulousness of the hands with mental and moral deterioration. The pupils are pinpoint. There is marked obstipation. Scars from infected hypodermic marks are visible on the forearms and the thighs. The continued pruritus leads to scratch marks, nose picking and chronic thickening of the skin.

WITHDRAWAL SYMPTOMS—The course of morphinism is punctuated by withdrawal symptoms best illustrated in the laboratory by the effect of the drug on the frog. The morphinized frog after several hours of depression develops irritability and spinal convulsions often leading to clonus and death. The morphinist after several hours of abstinence from the drug develops yawning, lacrimation and sneezing. Later he becomes nervous, restless and irritable. He develops abdominal and muscle cramps, tachycardia, diarrhea and sweating. There may be an elevation of temperature. In the absence of treatment he may become violent and maniacal or lapse into coma.

TREATMENT—The treatment of morphine addiction presents a problem similar to that of chronic alcoholism. The important difference is that morphinism is a Federal offense which by the Harrison Act the physician is required to report to the Federal authorities. At the present time chronic alcoholism does not come under Federal or even local police jurisdiction unless the welfare of individuals or the peace of the community is threatened.

The treatment of morphine addiction can rarely if ever be carried out except under institutional conditions. The physician should avoid those commercial enterprises which specialize in the management of addicts. Here the unfortunate victim meets with those who exploit drugs and will later induce him to return to his vice.

The institutions best adapted for the treatment of morphine addiction are the *Federal Farms* operated under the auspices of the United States Public Health Service and the *large psychiatric hospitals* privately or publicly managed that have full facilities for physical and mental rehabilitation.

The first aim of treatment is to withdraw the drug. The majority of experienced physicians favor gradual withdrawal by the use of decreasing dosages at increasing intervals. A minority including this author practice complete withdrawal or what the addict calls *cold turkey*.

During the period of withdrawal of the drug the patient must be properly treated with *sedatives*. Since very little food intake can be accomplished by the normal route, a *continuous intravenous injection* of 5 per cent dextrose in saline is highly desirable as soon as the patient can be controlled.

The *continuous warm bath* lessens the amount of sedative drug that has to be administered. Wherever possible reliance should be placed on *paraldehyde*. Highly agitated and maniacal patients may be mollified by the use of the drugs employed in basal anesthesia. In these cases *alvertin* with *amylene hydrate* is given by rectum. Two or three times the hypnotic doses of *barbiturates* are given orally or by intramuscular injection.

As soon as the patient has quieted down sufficiently the intravenous

STIMULANTS OF THE CENTRAL NERVOUS SYSTEM

Drugs may stimulate the central nervous system as a principal action a subsidiary manifestation or a toxic effect. *Xanthine derivatives* and *strychnine* produce cerebral stimulation as a primary effect. *Amphetamine*

TABLE 23—A COMPARISON OF MORPHINE AND DEMEROL

| | Demerol | Morphine |
|---------------------------------|---|--|
| Pain ¹ | 100 mg (1 $\frac{1}{4}$ gr) intramuscularly | 10 mg (1 $\frac{1}{2}$ gr) subcutaneously |
| Smooth Muscle ² | Relaxed | Stimulated |
| Colic | Relieved | Increased |
| Bronchospasm | Relieved | Increased |
| Angiospasm | Relieved | Increased |
| Gastric Dilatation | Not favored | Favored |
| Constipation | Not favored | Favored |
| Urinary Retention | Not favored | Favored |
| Pupils ³ | Unaffected | Contracted |
| vomiting ⁴ | Less than 10 per cent | More than 25 per cent |
| Respiratory Center ⁵ | Unaffected | Depressed |
| Cortical Action ⁶ | Euphoria | Depression |
| Cough Center | No effect | Depression |
| In Obstetrics | Favorable effects on mother and child | Respiratory depression to be feared in child |
| Combination with Scopolamine | Excellent | Excellent |
| Preoperative Use | Preferred | |
| Postoperative Use | Preferred | |

¹ Except for the more rapid relief of pain from an intravenous dose of morphine, demerol seems by all odds to be preferred as an analgesic. Certainly it should be tried first before resort to morphine, particularly in those instances in which the pain is accompanied by or due to muscle spasm.

² The comparative effects on smooth muscle indicate that demerol is to be preferred in the pain that is due to such diverse conditions as gastro-intestinal hypermotility, pylorospasm, sigmoid spasm, gallbladder and urinary colic, dysmenorrhea due to uterine cramps, dysuria due to peroneal spasm, angina pectoris, peripheral angiospasm and the dysenteries.

³ The normal size of the pupils with demerol is of assistance to the anesthetist who is not then deprived of the information obtained from the pupillary reflexes as in the case of the pinpoint pupils resulting from morphine.

⁴ The incidence of the untoward emetic effect is all in favor of demerol.

⁵ Respiratory center depression constitutes a toxic morphine effect unless there is hyperventilation. Under any other circumstance the negative effect of the demerol is advantageous.

⁶ The euphoria of demerol in contradistinction to morphine depression is of some significance in the analgesic effects on patients who are afflicted with inoperable carcinomata (p. 572) since from a psychological standpoint the sense of exhilaration is beneficial.

(Benzedrine, Dexedrine[®]) causes a more marked stimulation though its action is subsidiary to its effect on the involuntary nervous system. *Picrotoxin*, *camphor* and *metrazol* are employed almost wholly for analeptic effect in overcoming depression due to poisonings and for the therapeutically induced convulsions in the treatment of the psychoses. Therapeutic doses of *nikethamide* have a stimulant effect on the medulla.

atropine like action on the structures innervated by cholinergic nerves and a direct depression of the muscle fibers themselves. The segmental tone and contractions of the intestinal musculature are diminished or abolished but the propulsive action is not altered. Constipation does not result from the prolonged use of the drug in therapeutic amounts. The antispasmodic effect is noted in the uterus and on the bronchial musculature in asthma. Relaxation of the walls of the blood vessels may result in a sharp fall in blood pressure when the drug is given intravenously.

Miscellaneous Effects—Demerol produces some dryness of the mouth flushing of the face dizziness nausea and vomiting it has no action on cough.

Tolerance and Addiction—Demerol is rapidly destroyed by the liver and the effective dose can be repeated at 4 hour intervals prolonged use leads to the development of habituation particularly in individuals who

TABLE 200—THERAPEUTICS OF DEMEROL

| | |
|----------------------|---|
| Pain ¹ | Central analgesia as with opiate 50 mg ($\frac{3}{4}$ gr) of demerol the equivalent of 22 mg ($\frac{1}{4}$ gr) of codeine 100 mg ($1\frac{1}{4}$ gr) of demerol the equivalent of 10 mg ($\frac{1}{8}$ gr) of morphine |
| Smooth Muscle | Relaxed use demerol in dysmenorrhea (p 2561) for uterine effect in asthma (p 2101) for bronchial muscle effect, and in angiospasm (p 791) for depression of vessel wall |
| Preoperative Use | Substitute 50 to 100 mg ($\frac{3}{4}$ to $1\frac{1}{2}$ gr) for morphine in preoperative preparation may combine with scopolamine and/or barbiturate |
| Obstetric Anesthesia | Use 100 mg ($1\frac{1}{4}$ gr) with scopolamine as soon as pains become severe may require repeat in a few hours combination tends to shorten labor without adversely affecting baby |
| Postoperative Use | Excellent analgesic without hazard of favoring gastric dilatation ileus obstipation or urinary retention |

¹ See Footnote 1 to Table 223

were at one time morphine addicts. Dependence on demerol is difficult to produce in normal individuals addiction to demerol is not attended by severe withdrawal symptoms.

Demerol and Morphine—An attempted estimation of the comparative efficacy of demerol and morphine comes immediately to the mind of the pharmacologist and practitioner (Chart 22, p 3865)

TRICHLORETHYLENE

Trichlorethylene produces prolonged anesthesia of the fifth nerve following exposure by inhalation in industry. It has therefore been marketed in 1 cc sealed glass tubes for inhalation therapy in the treatment of trigeminal neuralgia (p 1482) and angina pectoris (p 890). The drug is administered with the patient recumbent. Addiction may be encountered. The results are more often disappointing than otherwise.

The diuretic efficacy of the xanthines is feeble compared to the effect produced by the mercurials (p 2257) Despite enthusiastic claims for aminophylline it is but slightly superior to the caffeine beverages

Effects on Circulation—The circulatory effects of therapeutic doses of the xanthines are irregular transitory and altogether of minor importance

Tracings may readily be obtained from isolated heart preparations that show a caffeine stimulation of the myocardium Other experimental records demonstrate a dilatation of the bed of the coronary vessels However it is a far cry from the effects of therapeutic doses given under clinical conditions to those produced by enormous doses administered under laboratory conditions

Despite the extensive use of caffeine as a circulatory stimulant particularly in acute failure and conditions associated with shock beneficial effects are not demonstrable in the human subject The cerebral stimulation adds anxiety to the rest of the clinical difficulties and augments the circulatory load

More tangible claims of efficacy are made for aminophylline which is said to increase cardiac output and respiratory volume while decreasing peripheral circulatory resistance

Effects on Smooth Muscle—The use of aminophylline has been advocated for its relaxation of smooth muscle This effect is purported to be of value in the treatment of hypertension vasospastic disorders the anginal syndrome and diseases of the coronary arteries (p 983)

After clinical trial the initial enthusiasm has given way to a profound skepticism Aminophylline has proven of value however when given intravenously in the treatment of bronchial asthma and spasm of the cerebral arteries and in relieving attacks of Cheyne-Stokes respiration by reducing venous and intrathecal pressures

Effects on Striated Muscle—In the laboratory animal caffeine produces a hypertonicity of striated muscle For this reason the use of the drug has been suggested in the treatment of fatigue and the muscular dystrophies It is however of little or no value

Antidotal Use of Caffeine—Caffeine or the caffeine beverages are favorite pharmacological antidotes in the treatment of poisoning due to the cerebral depressants The xanthine drugs are given to sufferers from acute and chronic alcoholism opiate poisoning and barbiturate bromide and chloral intoxications

It is questionable whether the caffeine antidotal effect is salutary Certainly the patient does not succumb to cortical depression in these poisonings The therapeutic desideratum is the maintenance of respiration This is not favored by the use of therapeutic doses of caffeine but is most easily accomplished by the institution of artificial respiration and the inhalation of oxygen Plying the victim with toxic doses of caffeine is an example of misdirected zeal

Toxicology—Caffeine poisoning from medicinal use is rare The toxicology of caffeine is mostly concerned with the abuse of the caffeine beverages It is not at all unusual for patients to admit drinking innumerable cups of tea or coffee and several to a dozen bottles of the carbonated cola beverages

XANTHINE DERIVATIVES

The most widely used stimulants of the central nervous system are the xanthines which include caffeine theobromine and theophylline

Caffeine Beverages—Most of the intake of caffeine and related substances is accomplished through the ingestion of coffee tea cocoa mate and the carbonated cola drinks

It is estimated that the annual per capita consumption of coffee in the United States exceeds 10 pounds The ordinary cup of coffee or strong tea contains 0.1 to 0.13 gm ($1\frac{1}{2}$ to 2 grains) of the alkaloid

The great dependence of many patients on their coffee quota makes it very difficult for the physician to interdict the use of the cup that cheers Coffee drinking is often prohibited in the management of gastroduodenal ulcer gout cardiovascular disease hypertension and the anginal syndrome It is my opinion that the discomfort of renouncing caffeine beverages is out of all proportion to possible benefits Unless the patient voluntarily states that coffee drinking is noxious and had better be abandoned I do not forbid its use for medical reasons

TABLE 224—COMMONLY USED XANTHINE PREPARATIONS

| Preparation | Average Dose |
|---|-----------------------------|
| Caffeine USP | 0.15 gm ($\frac{3}{4}$ gr) |
| Caffeine Citrate USP | 0.35 gm (5 gr) |
| Caffeine and Sodium Benzoate USP | 0.35–0.5 gm (5–7½ gr) |
| Theobromine N.N.R. | 0.35–0.5 gm (5–7½ gr) |
| Theobromine and Sodium Salicylate USP | 1 gm (15 gr) |
| Theobromine and Sodium Acetate USP | 0.5–1 gm (7½–15 gr) |
| Theocalcin N.N.R. | 0.5–1 gm (7½–15 gr) |
| Theophyllin USP | 0.2–0.35 gm (3–5 gr) |
| Theophylline and Sodium Acetate USP | 0.2–0.35 gm (3–5 gr) |
| Theophylline Ethylene Diamine USP (Aminophylline) | 0.24–0.48 gm (4–7 gr) |

Pharmacology and Therapeutics—The xanthines stimulate the central nervous system manifest a diuretic action on the kidney and relax smooth muscle

Stimulation of the Central Nervous System—The stimulant effect of caffeine manifests itself primarily on the *cerebral cortex* It is less manifest on the *medullary centers* and has an inappreciable effect on the cord This relative action is the opposite of strychnine which has its most potent effect on the cord and diminishes in intensity as the cortex is approached

Cerebral stimulation occurs with therapeutic doses of caffeine Theophylline and theobromine have relatively lesser effect

Stimulation of the medullary centers particularly that which controls respiration can be obtained only with doses that approach the toxic level Medullary stimulation is not within the range of the therapeutic accomplishment

Diuresis—The xanthines exert a true diuretic effect (p 2257) Theophylline theobromine and theocalcin are relatively more efficient than is caffeine Coffee diuresis is partially attributable to increased fluid intake In point of fact very little more is accomplished by the use of the relatively expensive compounds than can be obtained from the ingestion of the caffeine beverages

The bitter taste of *nux vomica* finds employment in the prescription of this preparation as a *stomachic*. Other bitters are to be preferred since they do not possess untoward side actions.

The administration of strychnine has been advocated in the treatment of morphine and barbiturate poisonings but we have no confidence in its antidotal effects and fear the hazard of adding another type of alkaloidal poisoning. It has been claimed also that strychnine is of value with or without quinidine (p 861) in the treatment of paroxysmal cardiac irregularities. Once again we find ourselves on the side of the skeptics and place our entire reliance on quinidine and the measures elsewhere described (p 873).

Poisoning—The widespread and injudicious use of strychnine results in the appreciable incidence of poisoning of varying degree. Perhaps the most common cause of severe poisoning is the inadvertent ingestion of gaily colored A.B.S. cathartic pills (N.F.) by young children.

The symptoms vary from a marked increase in the spinal reflexes to *tetanic convulsions* with *opisthotonos* that may prove fatal. Convulsions last upwards of one minute during which there is respiratory arrest. This is followed by a period of depression and another seizure ten or fifteen minutes later. Death may occur from exhaustion and anoxemia after two or more seizures.

Treatment—The treatment of strychnine poisoning is concerned primarily with lowering reflex excitability. The usual *gastric lavage* is deferred until the patient has been quieted. The *sedative drugs* employed are the soluble rapidly acting *barbiturates* (sodium amytal or sodium pentothal) given intravenously at a slow rate until the patient goes to sleep or convulsions cease. Usually 0.4 to 1.0 gm. (6 grains to 15 grains) of sodium amytal or 0.3 to 0.7 gm. (5–11 grains) of sodium pentothal suffice. If barbiturates are not at hand general anesthesia is induced with *chloroform* or *ether*.

With the patient quiet the physician may undertake gastric lavage with strong tea or potassium permanganate solution 1:1000. The strychnized patient does not vomit and lavage is necessary to assure the removal of ingested drug. Careful supervision is continued for many hours to resume prompt treatment of recurrent excitement or convulsions.

SYMPATHOMIMETIC AMINES

Sympathomimetic amines (p 3876) have their principal action on the involuntary nervous system. As a side effect however they may stimulate the cerebrum and in the instance of amphetamine sulfate this subsidiary action assumes primary importance.

AMPHETAMINE SULFATE (BENZEDRINE DEXEDRINE)

Amphetamine sulfate is a powerful analeptic drug which produces a true *cortical stimulation*. In states of severe or extreme fatigue it removes the desire for sleep and rest and substitutes the sensations of self confidence and well being. It improves the performance of skilled acts during fatigue states. The effects of the drug appear about one hour after oral ingestion and continue for six to eight hours. Single doses should not exceed 10 mg. ($\frac{1}{4}$ gr.) and should not be repeated later than noontime.

The toxic manifestations include restlessness irritability tremors of the fingers insomnia nausea vomiting indigestion pylorospasm palpitation and a general feeling of tenseness There may be noted a cardiac irregularity from premature contractions or a paroxysm of fibrillation

The objective findings in caffeine poisoning are rarely severe There may be a slight tachycardia and a negligible elevation of blood pressure These trivial phenomena attest the feebleness of the circulatory action of caffeine

The treatment of caffeine poisoning consists in the discontinuance of the use of the drug the substitution of a decaffeinated beverage for the coffee or tea and the use of mild sedatives such as the barbiturates

STRYCHNINE

Strychnine is the chief alkaloid of *nux vomica* the seeds of an Indian tree which also yield the alkaloid brucine The effects of the extract and tincture of *nux vomica* are due chiefly to the strychnine contained therein

The official preparations of *nux vomica* and strychnine include the pure alkaloidal salt *Strychnine Sulfate U.S.P.* usually administered in doses of 2 mg ($\frac{1}{30}$ grain) the *Extract of Nux Vomica* 15 mg ($\frac{1}{4}$ gr) and the *Tincture of Nux Vomica* 1 cc (15 minims)

Absorption and Excretion—Strychnine and *nux vomica* are rapidly absorbed from the gastro intestinal tract They are equally distributed to all tissues Approximately 20 per cent of the drug is excreted in the urine the rest being destroyed in the liver within ten hours

Pharmacology—The principal action of strychnine is stimulation of the central nervous system most marked in the cord and less intense in the medulla and the cerebrum

The cord effect consists in an increase of reflex excitability A minimal sensory impulse does not stop at the production of a discrete motor response but diffuses up and down the cord to produce a mass reaction leading to spinal convulsions Reciprocal inhibition of reflexes is lost The final effect is determined by the stronger muscle group so that the convulsion is symmetrical incoordinate and chronic resulting in a tetanus with opisthotonos (p 3520)

With larger doses there is some stimulation of the medullary centers including that of respiration With still larger doses there is excitation of the cerebral cortex and increased sensitivity of the organs of special senses The latter effects are not obtained until doses which are toxic for the cord have been reached

Therapeutics—Despite its potent pharmacological action strychnine is a useless drug but unfortunately one that is widely employed in clinical medicine

Strychnine is used as a cerebral stimulant to combat respiratory and circulatory failure to stimulate smooth and striated muscle as a cathartic and aphrodisiac to elevate blood pressure to increase the tonicity of the weakened and incontinent sphincters to increase the acuity of hearing and vision to improve nutrition to combat paresis and paralyses and to relieve shock

In none of these instances is the drug of the slightest value It may be noxious through its stimulation of the cord reflexes

Metrazol produces an intense stimulation of all parts of the central nervous system, particularly of the motor cortex. Its stimulant action on the medulla is more apparent in depressed than normal structures. It has been advocated as a respiratory and a circulatory stimulant but in our opinion, has only uncertain therapeutic value. It is our belief that metrazol has greater potential for harm than good and we reserve its use as a measure of desperation when all else has failed.

The central action of metrazol is used chiefly to produce convulsions in the treatment of psychoses (p. 1364). For this purpose 300 to 700 mg are given intravenously. Convulsions occur in a matter of 15-20 seconds and are typically epileptiform in nature. The convulsive state is followed by a period of confusion or amnesia lasting several hours. The production of metrazol convulsions is not without danger and has been attended by an appreciable incidence of fractures of the thoracic spine. The simultaneous use of *curare* (p. 3888) affords a means of preventing such trauma.

Toxicology—Overdosage with camphor or metrazol produces epileptiform convulsions followed by death from respiratory failure. Treatment is symptomatic using intravenous barbiturate.

NIKETHAMIDE (CORAMINE)

Nikethamide N.N.R. is a synthetic proprietary which is widely used as a cardiorespiratory stimulant. It is supplied in 25 per cent aqueous solution in ampoules of 1-2 cc.

Pharmacology—Coramine in therapeutic doses produces a well marked reflex stimulation of the respiratory center through the carotid sinus. Convulsions result from toxic doses by direct stimulation of the motor cortex. The effect on the respiratory center is more apparent when the structure is depressed by morphine, volatile anesthetics or barbiturates.

Therapeutics—Many anesthetists use coramine in the acute respiratory paralysis that occurs during the course of a general anesthesia. It has been advocated by its enthusiasts to increase coronary blood flow and directly stimulate heart muscle. There is no evidence, however, that these effects occur. Being an amide of nicotinic acid, coramine has proved effective in the treatment of pellagra (p. 624).

Following the administration of the drug in moderate doses about 10 per cent of persons experience palpitation a feeling of tension tachycardia and an increase in blood pressure

Therapeutics—Small doses of amphetamine sulfate (5 to 10 mg [$\frac{1}{2}$ to $\frac{1}{8}$ gr]), repeated at eight hour intervals may be used to combat fatigue and sleepiness especially in military operations The drug is useful in overcoming a *minor depression* and is of great value in the treatment of *narcolepsy* (p 1307) and *obesity* (p 697) In the latter instance the drug produces an anorexia Enthusiasts claim that amphetamine is of value in the prevention and treatment of *seasickness and airsickness*

Toxicity—Excessive doses of amphetamine produce headache anorexia abdominal cramps constipation dizziness irregular heart action palpitation tachycardia mental disturbances euphoria impaired judgment and hallucinations Vasomotor collapse and death have been reported Treatment involves cessation of the drug and the use of sedatives

PICROTOXIN

Picrotoxin is a powerful stimulant of the cerebrospinal axis It exerts its main action on the midbrain and medulla

Picrotoxin is not an official drug A 1:1000 solution may be improved with sterile isotonic saline solution and 20 cc ampoules containing 3 mg to the cubic centimeter are commercially available

Picrotoxin is used exclusively in the treatment of barbiturate poisoning (p 3842)

CAMPHOR AND METRAZOL

Camphor and a synthetic camphor like compound metrazol are powerful stimulants of the central nervous system They are used as medullary stimulants in poisonings and as convulsants

Preparations—*Camphor (U.S.P.)* is given orally in gelatin capsules *Camphor in Oil (N.F.)* is available in 20 per cent solution in ampoules for intramuscular injection the dose is 0.2 gm (3 gr) Other U.S.P. preparations *Camphor Liniment* *Spirit of Camphor* and *Camphor Water* are without important systemic action

Metrazol (N.N.R.) is available in aqueous solution for parenteral use Each cubic centimeter contains 100 mg ($1\frac{1}{2}$ grains) and tablets of 100 mg are prepared for oral use The accepted dosage is 100–300 mg ($1\frac{1}{2}$ – $4\frac{1}{2}$ gr) injected subcutaneously intramuscularly or intravenously and repeated as required

Pharmacology and Therapeutics—*Camphor* in large doses stimulates the central nervous system and may cause epileptiform convulsions In therapeutic doses the stimulation is negligible Subcutaneous and intramuscular injections of camphor in oil produce some medullary stimulation as a reflex response to the intense local irritation produced by the drug When applied to the skin locally camphor stimulates nerve endings sensitive to cold and produces a feeling of cold when vigorously applied as a liniment it is a good *rubefacient* In the stomach small doses are *carminative* The widespread use of camphor as a circulatory and respiratory stimulant is unsupported by any reliable clinical or laboratory evidence

The principal exceptions to the anatomic pharmacologic identity of the two divisions are (1) The sweat glands which are innervated by anatomic sympathetic fibers and which respond to acetylcholine and (2) the skeletal musculature which is innervated by the central nervous system and which responds to acetylcholine.

CHOLINERGIC DRUGS

The *craniosacral (vagal)* subdivision of the involuntary nervous system is stimulated pharmacologically by (1) supplying the chemical medi-

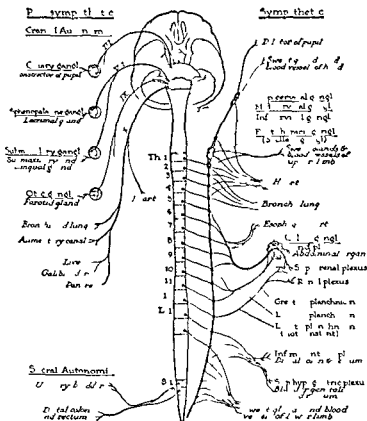


Fig 1127—Scheme of autonomous system

ator *acetylcholine* and (2) by inhibiting the *cholinesterase* by which *acetylcholine* is destroyed or inactivated

The available drugs which supply the mediator include *acetylcholine*, *acetyl β -methyl choline* (*mecholyl*), *carbaminoylcholine* (*doryl*) and *pilo carpine*. The substances which inhibit or destroy cholinesterase are *physo stigmine* (*eserine*) and *prostigmine*.

Toxicology—As a result of the widespread vasodilatation and cardiac arrest the cholinergic drugs may produce nausea vomiting circulatory

CHAPTER 180

PHARMACOTHERAPY DRUGS WITH PRINCIPAL EFFECTS ON THE INVOLUNTARY NERVOUS SYSTEM

The Cholinergic Drugs

Acetylcholine
Mechoyl
Doryl
Pilocarpine
Physostigmine
Neostigmine

Depressants of the Cholinergic System

Belladonna
Atropine
Scopolamine
Homatropine
Eucatropine
Novatropine
Syntropan
Trasentun

The Adrenergic Drugs

Epinephrine
Ephedrine
Amphetamine
Neosynephrine
Propadrine
Paredrine
Privine
Kephrine

Inhibitors of Adrenergic Effects

Ergotamine
Ergotoxine
Nicotine

Ganglionic Depressants

Tetra-ethylammonium Compounds
(p 1007)

The involuntary nervous system has distinct anatomic physiologic and pharmacologic characteristics. From the standpoint of drug therapy, the cholinergic and adrenergic subdivisions are clearly defined. The two divisions reciprocally innervate end organs and tend to antagonize each other. From a functional standpoint, this type of innervation is particularly useful in accomplishing physiologic changes. The chemical mediators of one system produce effects resembling the inhibition or destruction of the other.

Cholinergic Division—The cholinergic division is synonymous with the anatomic craniosacral and the physiologic parasympathetic division of the autonomic system. Stimulation of these nerves is attended by the liberation of *acetylcholine*, a chemical mediator which acts on glands, smooth muscle, autonomic ganglia and skeletal muscle. This substance is destroyed or inactivated by an enzyme, *cholinesterase*. Moreover, its action is inhibited or blocked by *atropine* (on smooth muscle or glands) and by *nicotine* and *curare* (on ganglia and skeletal muscle). Consequently, the effects of cholinergic stimulation may be accomplished by the administration of acetylcholine or one of its derivatives and by inhibiting the action of cholinesterase.

Adrenergic Division—The adrenergic division is the thoracolumbar or sympathetic division of the autonomic system. Stimulation of the postganglionic fibers of this division results in the liberation of *sympathin*, an epinephrine-like substance, and *epinephrine* (from the adrenal medulla). The action of sympathin is limited in all likelihood by its enzymic destruction by *aminoxidase*. It is blocked by *ergotoxin* and certain diuretic derivatives.

The treatment of poisoning with the cholinergic drugs is the rapid administration of atropine sulfate (0.001 gm [$\frac{1}{60}$ grain]) The cholinergic drugs should not be administered unless a hypodermic syringe loaded with atropine is available for immediate intravenous injection

DEPRESSANTS OF THE CHOLINERGIC SYSTEM

The cholinergic depressants produce a pharmacologic effect that is opposite to that of the drugs previously described

The principal cholinergic depressants are *belladonna* and the natural and synthetic preparations of similar nature As with the cholinergic drugs these substances have a qualitatively similar effect but there are marked quantitative differences necessitating a careful choice of preparation depending upon indication

Hyoscyamus and *stramonium* have been virtually abandoned in general clinical practice *Stramonium* is still used in the so called asthma cigarettes *Tincture of Hyoscyamus* is employed by the urologist to relieve urinary tenesmus

TABLE 227—PREPARATIONS AND DOSAGES OF DEPRESSANTS OF THE CHOLINERGIC SYSTEM

| | Average Dose |
|--|---------------------------------------|
| Extract of Belladonna, USP | 15 mg ($\frac{3}{4}$ gr) |
| Tincture of Belladonna, USP | 1 cc (15 minims) |
| Atropine Sulfate, USP | 1 mg ($\frac{1}{300}$ gr) |
| Scopolamine Hydrobromide USP (Hyscine) | 0.5 mg ($\frac{1}{200}$ gr) |
| Homatropine Hydrobromide USP | 1 cc |
| Eucatropine Hydrochloride N.R. | 5-10% |
| Novatropine N.R. | 2.5 mg ($\frac{1}{40}$ gr) |
| Syntropan N.R. | 50 mg ($\frac{3}{4}$ gr) (tablets) |
| | 10 mg ($\frac{1}{30}$ gr) (ampoules) |
| | 75 mg ($1\frac{1}{4}$ gr) (tablets) |
| | 75 mg in 1.5 cc (ampoules) |
| Trasentan | |

Pharmacology—The belladonna drugs act centrally and peripherally The central action consists of stimulation of the cerebrum and the medullary centers These effects most often result from idiosyncrasy and poisoning

Peripherally the belladonna derivatives depress or paralyze the structures innervated by the postganglionic fibers of the cranio sacral or vagal division of the involuntary nervous system

Toxicology—Atropine poisoning usually occurs from idiosyncrasy less often from overdosage It is fairly frequent among infants and young children receiving the drug

Atropinism is characterized by dryness of the mouth thirst mydriasis with blurring of vision cutaneous flush fever tachycardia restlessness muscle incoordination and symptoms of a toxic psychosis (belladonna jag)

The period of central stimulation is followed by extreme depression collapse and respiratory paralysis The fatal dose varies from 10 to 100 mg ($\frac{3}{8}$ to $1\frac{1}{2}$ gr)

Treatment consists of gastric lavage with strong tea sedation and the administration of pilocarpine in repeated 2 mg doses until salivation re-

collapse and syncope Excessive doses or the average dose in a sensitive individual may cause a marked fall in blood pressure and cardiac arrest

TABLE 225—PREPARATIONS AND DOSAGES OF THE CHOLINERGICS

Acetylcholine N N R

Use mecholyl

Mecholyl (Acetyl beta methylcholine)

Tablets 0.2 gm (3 gr) ampoules 0.025 gm ($\frac{3}{8}$ gr)

Mecholyl may be given by iontophoresis (p 000) in the treatment of indolent ulcers myopathies arthropathies peripheral angiospasm and gangrene

When mecholyl is given by hypodermic the pharmacologic antidote atropine must be held in readiness for immediate use if necessary

Doryl (Carbaminoethylcholine)

Ampoules 1 cc = 0.25 mg tablets 2 mg ($\frac{1}{2}$ gr)

Pilocarpine Hydrochloride NF

Tablets 5 mg ($\frac{1}{4}$ gr) as miotic 1 to 5 per cent solution

Physostigmine Salicylate USP

Tablets 1 and 2 mg ($\frac{1}{64}$ and $\frac{1}{8}$ gr) as miotic 0.1 to 1 per cent solution

Neostigmine Methyl Sulfate N N R

Ampoules 1:2000 and 1:4000

Neostigmine 1:4000 is best used in the prevention of postoperative ileus The 1:2000 solution is reserved for active treatment

* The cholinergics are contraindicated in spasmodic asthma and in threatened pulmonary edema

TABLE 226—PHARMACOLOGY AND THERAPEUTICS OF THE CHOLINERGICS

For miosis and reduction of intra ocular tension

Use 1 to 5 per cent pilocarpine nitrate or 0.1 to 1 per cent physostigmine salicylate (eserine)

Most useful in the prevention and treatment of glaucoma (p 1578)

To increase local vascularity and relieve angospasm

Mecholyl by iontophoresis (p 3873)

To restore sinus rhythm in paroxysmal irregularities (p 873)

Mecholyl subcutaneously in dose of 20 to 30 mg ($\frac{1}{2}$ to $\frac{3}{4}$ gr)

To relax smooth muscle spasm and stimulate peristalsis

In retention of urine use doryl (1 cc = 0.25 mg)

In ileus use neostigmine methyl sulfate 1 cc of 1:2000 or 1:4000 solution

Do not use doryl and neostigmine at the same time Injections should be at least 2 hours apart and may be repeated at 4 or 6 hour intervals Cholinergics are preferred to posterior pituitary solutions which have undesirable vasopressor effects

To stimulate striated muscles.

In myasthenia gravis (p 2886) use neostigmine

Simultaneously an attack of bronchial asthma may be simulated by the intense bronchospasm associated with bronchorrhea

The cholinergic drugs are used with caution in allergic individuals and those with abnormalities of vascular tension whether hypertension or hypotension

TABLE 225—ADRENEPGIC DRUGS

Epinephrine Hydrochloride U.S.P.

1:10,000 aqueous solution in ampoules 1:1000 aqueous solution in ampoules and brown bottles 1:100 aqueous solution for spray 1:500 in oil in ampoules for intramuscular injection

Epinephrine in 1:100 spray preferred for atomizer use in asthma (p 2101) (1:500 solution in oil best for prolonged action 1:10,000 concentration increases local cocaine effect by delaying absorption through vasoconstriction)

Ephedrine Hydrochloride U.S.P.

Capsules 15 and 30 mg ($\frac{1}{4}$ and $\frac{1}{2}$ gr) for oral use 0.5–4 per cent solution for local and systemic use 0.5 per cent nasal jelly

Ephedrine and propadrine capsules with 15 mg ($\frac{1}{4}$ gr) of phenobarbital are preferred for oral use in milder allergies such as vasomotor rhinitis (p 2097)

Amphetamine Sulfate N.N.R. (Benzedrine)

1 per cent solution for local and systemic use Inhaler for vasomotor rhinitis (p 2097) tablets 5 and 10 mg ($\frac{1}{12}$ and $\frac{1}{6}$ gr) for oral use

Amphetamine inhalant offers rapid and convenient vasoconstriction but frequent or sustained use may damage membrane and lead to intractable epistaxis (p 2125) benzedrine tablets widely employed for "pick up" and "pep" particularly in narcolepsy fatigue and mild depressions also used to produce anorexia in treatment of obesity (p 697)

Neosynephrine N.N.R.

0.25 per cent aqueous solution for local use 0.25 per cent mineral oil emulsion 0.5 per cent nasal jelly 1 per cent aqueous solution for systemic use also mydriatic

Weak neosynephrine and prinine solutions preferred for nasal instillations and sprays

Paredrine or neosynephrine is preferred as mydriatic since it does not increase intra-ocular tension as do homatropine and related products

Propadrine N.N.R.

Capsules 24 and 45 mg ($\frac{3}{8}$ and $\frac{1}{2}$ gr) for oral use 1 per cent aqueous solution for local and systemic use 1 per cent nasal jelly

Ephedrine and propadrine capsules with 15 mg ($\frac{1}{4}$ gr) of phenobarbital are preferred for oral use in milder allergies such as vasomotor rhinitis

Paredrine

1 per cent aqueous solution for local and systemic use also mydriatic

Paredrine or neosynephrine is preferred as mydriatic since it does not increase intra-ocular tension as do homatropine and related products

Piprine

0.1 per cent aqueous solution for local use

Weak neosynephrine and prinine solutions preferred for nasal instillation and sprays

Kephrene Hydrochloride N.N.R.

Powder and rectal suppositories for local use

Kephrene powder most useful as styptic.

For treatment of shock, postural hypotension and carotid sinus syncope a 1 per cent aqueous solution of neosynephrine amphetamine propadrine or paredrine may be injected subcutaneously in 1 cc dose Pressor reaction less violent and more sustained than with epinephrine Useful also in spinal anesthesia to combat hypotension

Epinephrine U.S.P.—Epinephrine the hormone of the adrenal medulla bears a close resemblance to *sympathin* the hypothetical chemical mediator released after the stimulation of the adrenergic nerves Its important

TABLE 228—PHARMACOLOGY AND THERAPEUTICS OF CHOLINERGIC DEPRESSANTS

For Mydriasis

Use 1 per cent homatropine hydrobromide or 5 per cent eucatropine to produce pupillary dilatation (p 1533)

Mydriasis causes increased intra-ocular tension and may precipitate an acute glaucoma. Prevent by instillation of 1 per cent physostigmine salicylate as miotic or by substitution of adrenergic drug such as neosynephrine (p 3877)

To Relieve Travel Sickness

Sea air train and car-sickness best prevented and controlled by 0.5 mg (gr $\frac{1}{20}$) hyoscine orally or 0.65 mg (gr $\frac{1}{100}$) by suppository two hours before departure repeated every three hours for three doses if needed

For Cortical Amnesia

Scopolamine, with morphine or demerol produces twilight sleep in obstetrical and surgical anesthesia (p 3913)

Because of its amnesic effect scopolamine is greatly to be preferred to atropine for routine use. Combined with morphine or demerol there is often a highly gratifying effect suggesting a potentiation

For Medullary Stimulation

Despite wide use of atropine for respiratory center stimulation particularly to offset morphine effects therapeutic doses have little or no efficacy

To Relieve Heart Block and Carotid Sinus Syncope (p 922)

Atropine 1 mg ($\frac{1}{64}$ gr) by hypodermic

To Dry Oral Nasal and Bronchial Secretion

Atropine and scopolamine used preoperatively for this purpose also in early stages of coryza (p 2114)

To Relax Bronchial Muscle

Atropine best in asthma (p 2101)

Occasional relief from smoking asthma powder or cigarettes containing stramonium

To Relax Spasm in Gut Urinary Tract and Uterus

Oral doses of novatropine 2.5 mg ($\frac{3}{8}$ gr) syntropan 50 mg ($\frac{3}{4}$ gr) and traseptin 75 mg ($1\frac{1}{8}$ gr) most satisfactory

These products produce more favorable response if combined with 15 mg ($\frac{1}{4}$ gr) of phenobarbital. Particularly valuable in pylorospasm (p 1769) spastic constipation (p 1773) and dysmenorrhea (p 2561)

As Mechoyl Antidote (p 3873)

Use atropine 1 mg ($\frac{1}{64}$ gr) intravenously if necessary

To Relieve Tremor

Use scopolamine preferably in paralysis agitans (p 1505). Bulgarian belladonna is not superior

Optimum results if member of household can be taught to give small doses (0.3 mg [$\frac{1}{40}$ gr]) by hypodermic twice or thrice daily

turns. Should respiratory depression supervene *artificial respiratory measures* are indicated. Because of the rapid elimination of the drug recovery usually occurs

ADRENERGIC DRUGS

The sympathomimetic amines are available for local application or systemic use by oral and parenteral introduction. Their pharmacologic reactions differ quantitatively in certain instances so that each product may have advantages over others in the group or disad-

TABLE 229—ADRENERGIC DRUGS

Epinephrine Hydrochloride U.S.P.

1:10,000 aqueous solution in ampoules 1:1,000 aqueous solution in ampoules and brown bottles 1:100 aqueous solution for spray 1:500 in oil in ampoules for intramuscular injection

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Neosynephrine N.N.R.

0.25 per cent aqueous solution for local use 0.25 per cent mineral oil emulsion 0.5 per cent nasal jelly 1 per cent aqueous solution for systemic use also as mydriatic

Weak neosynephrine and privine solutions preferred for nasal instillations and sprays

Paredrine or neosynephrine is preferred as mydriatic since it does not increase intra-ocular tension as do homatropine and related products

Propadrine N.N.R.

Capsules 24 and 45 mg ($\frac{3}{8}$ and $\frac{3}{4}$ gr) for oral use 1 per cent aqueous solution for local and systemic use; 1 per cent nasal jelly

Ephedrine and propadrine capsules with 15 mg ($\frac{1}{4}$ gr) of phenobarbital are preferred for oral use in milder allergies such as vasomotor rhinitis

Paredrine*

1 per cent aqueous solution for local and systemic use; also mydriatic.

Paredrine or neosynephrine is preferred as mydriatic since it does not increase intra-ocular tension as do homatropine and related products

Privine*

0.1 per cent aqueous solution for local use

Weak neosynephrine and privine solutions preferred for nasal instillation and sprays

Kephadrine Hydrochloride N.N.R.

Powder and rectal suppositories for local use.

Kephadrine powder most useful as styptic.

* For treatment of shock, postural hypotension and carotid sinus syncope a 1 per cent aqueous solution of neosynephrine, amphetamine, propadrine or paredrine may be injected subcutaneously in 1 cc dose. Pressor reaction less violent and more sustained than with epinephrine. Useful also in spinal anesthesia to combat hypotension.

Epinephrine U.S.P.—**Epinephrine**, the hormone of the adrenal medulla, bears a close resemblance to *sympathin*, the hypothetical chemical mediator released after the stimulation of the adrenergic nerves. Its important

pharmacological effects are identical with adrenergic stimulation. They result from a direct action of the hormone on effector cells and are elicited on denervated structures.

Extirpation of the adrenal medulla produces no serious functional changes in the organism and the precise physiological role of epinephrine remains unknown. According to Cannon epinephrine aids in the preparation of the organism for emergencies requiring extreme physical effort.

Source Chemical Structure Properties—Epinephrine was the first hormone to be isolated in pure form to have its exact chemical structure determined and to be synthesized. It has served as the prototype of a long series of synthetic drugs, the *sympathomimetic amines*.

The adrenal medulla contains 1 to 3 mg of epinephrine per gram of tissue. The presence of the hormone in adrenal tissue is indicated qualitatively by the application of ferric chloride which gives a green color (*Vulcan reaction*) or of chromic acid which produces a brown color. The natural hormone is levorotatory and is about twice as active as the synthetic product which is racemic. Dextrorotatory epinephrine is inert.

Epinephrine is obtained from adrenal glands by extracting with dilute acid and precipitating the free alkaloid with ammonia after the precipitation of protein with alcohol. Chemically, the epinephrine molecule is composed of *cathechol* (dihydroxybenzene) and a complex amino alcohol (methylaminoethanol). Unlike other alkaloids it is sparingly soluble in alcohol and is not affected by alkaloidal precipitants. It is easily oxidized in neutral and alkaline solutions. It readily forms water soluble acid salts. Solutions of the salts are very unstable and decompose on exposure to air and light. On oxidation in alkaline solutions the colorless compound changes to pink, red and finally brown.

Epinephrine forms colored solutions as a result of many chemical reactions. These are the basis of several colorimetric methods of assay which allow the detection of epinephrine in a concentration of 1 in 50 million.

Since epinephrine is a vasoconstrictor intravenous injection of the drug produces a sharp rise in systolic and diastolic blood pressures which is transient and which varies with the dose. At the height of the pressor response there is a *reflex (vagal) bradycardia*. As a rule the hypertension gives way to a relative hypotension which is due to the weaker but more protracted inhibitory (vasodilator) action of epinephrine. This latter action may be elicited alone by the administration of minute doses or after *ergotoxin* which blocks the motor (vasoconstrictor) but not the inhibitor (vasodilator) effects in susceptible species (cat, dog).

ACTION ON SMOOTH MUSCLE OTHER THAN ARTERIOLAR—Epinephrine is a powerful dilator of the smooth muscle of the bronchioles and produces prompt relaxation when these structures are in spasm. The bronchioles are very sensitive to epinephrine and respond to doses with minimal effects on heart and arterioles. This action of epinephrine is extremely valuable for the relief of *bronchial asthma* (p. 2101).

Epinephrine in doses which cause marked cardiovascular responses relaxes the smooth muscle of the greater part of the gastro intestinal tract. The sphincters however react in a variable manner depending on their functional activity. As a rule they are contracted.

Epinephrine causes the smooth muscle of the *spinal tract* and thus causes an increase in the circu-

action is made use of clinically in the *Ascoli treatment* of malaria where increasing doses of the drug are given to empty the splenic reservoir of malarial parasites

Epinephrine relaxes the detrusor muscle of the urinary bladder but contracts the trigone and the sphincter. The human uterus is always contracted by the drug

Epinephrine has no demonstrable effect on the iris when instilled into the conjunctival sac. However on intravenous administration it causes a transient mydriasis which is more intense and prolonged on the denervated iris. The action is a direct one on the dilator muscle of the pupil

METABOLIC EFFECTS—Epinephrine increases the oxygen consumption of the tissues by a direct action on the cells which stimulates their metabolic processes. In man it raises the basal metabolic rate. The respiratory quotient rises with the increase in carbohydrate metabolism. There may be an increase in body temperature

The hormone increases hepatic glycogenesis causing hyperglycemia and glycosuria in direct antagonism to insulin. It also reduces the glycogen stores of skeletal muscle. The hyperthyroid subject is extremely sensitive to the effects of epinephrine and some of the symptoms of a thyrotoxic crisis are attributed to the participation of the adrenal medullary factor

Therapeutic Uses Preparations and Dosage **ALLERGY**—Epinephrine is of prime usefulness in the symptomatic treatment of allergic disorders particularly *bronchial asthma urticaria angioneurotic edema* and *serum reactions*. Since absorption occurs only from parenteral injection sites the hormone must be given subcutaneously intramuscularly or rarely intravenously. The preparation of choice is Solution of Epinephrine Hydrochloride U.S.P. a 1:1000 dilution in ampoules or sterile rubber capped vials. The average subcutaneous dose for adults varies from 0.2 to 0.5 cc (3 to 7 minims) which may be repeated if necessary when the initial dose fails to bring relief after fifteen minutes. For prolonged effect epinephrine in oil is administered intramuscularly in doses varying from 0.75 to 1.5 cc (12 to 22 minims). The effect generally lasts eight to sixteen hours so that the dose should not be repeated within that time. Patients with hypertension or other severe vascular disease should not receive this type of therapy. An occasional allergic individual may be sensitive to the peanut oil used as a solvent for the epinephrine. Thus the oil itself may produce an asthmatic seizure

Asthmatic patients often obtain considerable relief by inhalation of vaporized epinephrine directly into the lungs. For this purpose a more concentrated solution of the hormone (Solution of Epinephrine Hydrochloride 1:100 N.N.R.) is placed in a special nebulizer which delivers a finely dispersed spray. The nebulizer operated like an ordinary atomizer is fitted with a mouthpiece through which the patient inhales the vapor each time the bulb is compressed

An occasional patient with a severe case of asthma may require epinephrine intravenously. This is given by dissolving 1 cc of a 1:1000 solution in 300 cc of 10 per cent glucose (1:300,000) and then allowing the solution to run into a vein by gravity. Patients who are refractory to epinephrine when given by other routes will often respond to this type of therapy

The solution ready for injection should always be available

during skin testing administrations of serum or desensitizing injections in allergic individuals in order to avert the catastrophe of severe constitutional reaction. The mild irritation of a positive conjunctival test for serum sensitivity may be readily relieved by the instillation of a few drops of the 1:1000 solution of epinephrine into the conjunctival sac.

LOCAL VASOCONSTRICTION—Rhinologists employ epinephrine as a decongestant in the nose preliminary to intranasal surgery or manipulation, or simply as a spray for the relief of nasal obstruction in rhinitis or sinusitis. Since the initial vasoconstriction generally gives way to vaso-dilatation epinephrine is only feasible if it is combined with a more prolonged constrictor. For this purpose cocaine hydrochloride finds favor in rhinological practice. A suitable mixture consists of equal parts of 4 per cent cocaine hydrochloride solution and 1:1000 epinephrine hydrochloride solution.

Epinephrine should be combined with injectable local anesthetics. By causing local vasoconstriction it prevents systemic absorption of the drug, prolongs the anesthesia and minimizes the danger of systemic toxic effects. Concentrations of 0.02 to 0.05 mg. of epinephrine per cc. are used for this purpose.

As a hemostatic epinephrine has limited use because of its temporary effect. However it is occasionally combined with pressure hemostasis in nasal or oral bleeding.

CARDIAC ACTION—Epinephrine has been somewhat overrated as a therapeutically useful cardiac stimulant. Although an occasional moribund individual is apparently resuscitated by intracardiac or intravenous injection of the drug the dangers of producing ventricular fibrillation should make the physician hesitate before using this potent substance.

Toxicity—Small doses of epinephrine cause anxiety, tremor, restlessness and palpitation in a small group of normal individuals who are extremely sensitive to the drug (*sympatheticotonia*). This type of reaction is common in patients with exophthalmic goiter.

The inadvertent injection of a moderate dose intravenously may produce a serious cardiovascular accident in middle-aged individuals. Cerebral hemorrhage and thrombosis have been observed in such individuals.

During surgical anesthesia and in patients with heart disease epinephrine causes arrhythmias and may precipitate the onset of a fatal ventricular fibrillation. In cases of traumatic and medical shock (p. 908) epinephrine is contraindicated as it tends to accentuate the capillary defect by intensifying arteriolar constriction and local anoxemia. Patients with cocaine poisoning present many of the clinical features of epinephrine toxicity. The same symptom complex occurs in extreme force in patients with a *pheochromocytoma* of the adrenal medulla (p. 1264).

Ephedrine—Ephedrine is a natural alkaloid isolated from *ma huang*, a Chinese plant. Although it has been synthesized the main supply is from natural sources.

Chemical Structure and Properties—Ephedrine has the same basic structure as epinephrine but lacks the hydroxyl groups on the benzene ring and a three carbon atom aliphatic chain. This difference is responsible for its greater stability and the actions on the central nervous system. The alkaloid is levorotatory and is soluble in water and *anic* solvents.

It readily forms salts with mineral acids which are water soluble and whose water solutions are stable in the presence of air heat or light

Preparations and Dosage—Ephedrine is readily absorbed from the intestine and from parenteral sites The average oral dose is 15 to 50 mg ($\frac{1}{4}$ to $\frac{3}{4}$ gr) of the sulfate repeated at three to four hour intervals

The drug may be given as a hydrochloride or sulfate (both USP preparations) either in capsules tablets or ampoule solutions for subcutaneous injections Hypodermic tablets are also obtainable

For local applications the drug is available in 0.5 to 4 per cent solution either oily aqueous or isotonic (sodium chloride) Except for mydriasis 0.5 to 2 per cent solutions are generally adequate

Pharmacology—Ephedrine resembles epinephrine pharmacologically in many respects but has several important differences Its important actions include *peripheral vasoconstriction* of the arterioles of the skin splanchnic areas and spleen It causes a *relaxation* of the bronchial muscles mydriasis and *stimulates* the myocardium and the conduction system When applied locally to mucous membranes it causes *constriction* of the capillaries which is followed by *vasodilatation* In addition to its peripheral actions which mimic those of epinephrine it *stimulates* the cerebrum and the medullary vegetative centers The latter action is responsible for its use as an *analeptic* Central stimulation results in *restlessness* and in *insomnia* which require barbiturates for control

Aside from its central action ephedrine differs from epinephrine in the following characteristics (1) it is more stable (2) it is effective after gastro intestinal administration (3) it possesses no action on denervated structures (4) it is not potentiated by cocaine and (5) there is no ergo toxin reversal

Its mode of action is not definitely known but the most plausible theory is that the drug prevents the rapid destruction of *sympathin* by amine oxidases in a manner analogous to the protection afforded acetylcholine by physostigmine

Therapeutics—Ephedrine in the form of its salts finds frequent use in *bronchial asthma* generally in prophylactic maintenance doses of about 25 mg ($\frac{3}{8}$ grain) several times daily *Hay fever* sufferers occasionally benefit from the systemic use of the drug

In conjunction with spinal anesthesia ephedrine combats the tendency to *hypotension* It is of some use in chronic *hypotensive states* and in *postural hypotension* (p 916) In *Stokes Adams syndrome* maintenance doses may diminish the frequency of seizures and increase the ventricular rate

Ephedrine may be used as an *antidote* for poisoning by depressant drugs such as morphine or the barbiturates It was the drug of choice in *narcolepsy* and *myasthenia gravis* before the advent of benzedrine and prostigmine In *enuresis* the increased vesical sphincter tone following the administration of ephedrine sometimes affords relief from the urinary incontinence

The central effects of ephedrine as mentioned above are sometimes sufficient to make its use for other purposes unpleasant To avoid these many of the pharmaceutical houses prepare combinations of ephedrine and small doses of various barbiturates in single capsule form

Ephedrine solution finds wide application as a *nasal mucosal decongestant* in ordinary colds sinusitis and allergic disorders. The watery solutions are preferable to those in oil because of the danger of lipid pneumonia from aspiration of the latter and the possibility that oil interferes with the action of the mucosal cilia. Isotonic solutions are theoretically less irritating than the simple aqueous preparations although actual differences have not been demonstrated in practice as yet.

Ephedrine may be applied to the nose in the form of drops spray or jelly.

Amphetamine (Benzedrine)—Amphetamine is a synthetic sympathomimetic amine resembling ephedrine chemically and pharmacologically.

Pharmacology—The mode of action of amphetamine is not clearly understood although some evidence suggests that like ephedrine it works by preventing the destruction of normally secreted sympathin. In general it stimulates adrenergic innervated structures causing peripheral constriction of arterioles and capillaries mydriasis cardiac stimulation and bronchial muscle relaxation.

Amphetamine causes *central nervous system stimulation* more marked than any of the other adrenergic drugs (p. 3876). It is a powerful *central respiratory stimulant* which makes it useful in respiratory depressions due to drugs. Its bronchodilator effect is however too slight for the relief of asthma. The cardiovascular responses are variable pressor effect generally being noted only after large doses.

On the *gastro intestinal tract* the effect of amphetamine is difficult to foretell in any given case. The stomach musculature after preliminary stimulation undergoes relaxation. The colon is frequently relaxed while effects on the small bowel are not predictable.

Locally in the eye amphetamine is a satisfactory short acting mydriatic. Applied to congested nasal mucous membrane it produces a marked vasoconstrictor effect not succeeded by dilatation as in the case of epinephrine.

The drug is readily absorbed from the gastro intestinal tract subcutaneous or intramuscular sites. The amount absorbed from mucous membrane surfaces is ineffective systemically. **Excretion** occurs via the kidney.

Toxicity—Central stimulation including restlessness tremor confusion hallucinations hypertension hypotension palpitation vomiting and diarrhea have been noted. Habituation may occur in normal individuals who take the drug as a pickup.

Neosynephrine—Neosynephrine is a sympathomimetic amine whose effects resemble but are more prolonged than those of epinephrine. No central nervous action comparable to those of amphetamine or ephedrine has been observed. Chemically it is very similar to epinephrine the only difference being a single hydroxyl on the benzene ring in the meta position.

Uses Administration and Dosage—The drug is used as a nasal decongestant in colds sinusitis allergic disorders. For this purpose Neosynephrine Hydrochloride N. N. R. is available in $\frac{1}{4}$ and 1 per cent solutions Mineral Oil Emulsion (0.25 per cent) and jelly base (0.5 per cent). It must be kept in a dark bottle or atomizer without metal parts because metal and light tend to cause discoloration of the solution and the development of a most offensive odor. If used repeatedly in the . . . nt of nasal . . .

congestion the drug causes a paradoxical increase in congestion. It should therefore not be used habitually.

Because of its prolonged vasopressor action and the absence of cerebral stimulation, neosynephrine is of value as an adjunct in preventing vasomotor collapse in spinal anesthesia. The usual dose is 5 mg repeated if necessary given hypodermically in a 1 per cent sterile isotonic solution marketed for this purpose. In emergencies intravenous administration can be employed. Neosynephrine may also be combined with local anesthetics for local vasoconstriction. The concentration obtained from adding 5 to 4 drops of the 1 per cent solution to 10 cc of a 2 per cent procaine hydrochloride solution is generally sufficient.

Propadrine.—Propadrine is chemically and pharmacologically similar to ephedrine but causes less central stimulation. It has been accepted in N.N.R. for marketing in 50 and 24 mg ($\frac{3}{4}$ and $\frac{1}{2}$ grain) capsules, 1 per cent aqueous solution and as a nasal jelly. Its uses and dosage are similar to those of ephedrine.

Paredrine.—Paredrine is outstanding because of its *mydriatic effect*. A 1 per cent solution generally results in adequate pupillary dilatation in forty-five minutes which persists for only two hours. It is also useful as a nasal decongestant. The drug also has chemically useful *pressor action* and has been employed in spinal anesthesia for this purpose. A recent report suggests that it is beneficial in *Stokes Adams disease*. Central nervous system stimulation does not occur. Neither paredrine nor the closely related substance paredrinol have as yet been accepted by N.N.R.

Privine.—The use of privine is limited to local application as a nasal decongestant. It may be applied in 0.1 and 0.05 per cent solutions with minimum irritation of the inflamed mucous membrane.

Kephrine.—Kephrine is a sympathomimetic amine, a ketone precursor of epinephrine. It is used clinically as a styptic—and is available as kephrine Hydrochloride N.N.R. in powder and rectal suppositories.

INHIBITORS OF ADRENERGIC EFFECTS

Adrenergic effects are modified by ergotamine, ergotoxine and nicotine only; ergotamine has therapeutic usefulness.

Ergotamine and Ergotoxine.—Ergotamine and ergotoxine are derivatives of ergot (p. 2009). They act directly on cells innervated by adrenergic nerves and prevent stimulation by neurogenic mechanisms and by epinephrine. The epinephrine inhibition is known as the *Dale reversal*. Other drugs including the *dioxane derivatives* have similar properties but they are mere pharmacologic curiosities at the present time and do not need further discussion.

Ergotoxine has no present clinical function but ergotamine is widely used in *migraine* (p. 1506) and in *obstetrics* (p. 2617). The pharmacologic details are elsewhere described.

Nicotine.—Nicotine has pharmacologic but little therapeutic significance. The clinician's main concern relates to its effects in smoking.

Pharmacology.—Nicotine produces an initial stimulation followed by a depression of the cells of the autonomic ganglia, the central nervous system and voluntary muscle. Its central action may produce convulsions, vomiting and hyperventilation later followed by respiratory paralysis. The

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Pharmacology—Nicotine produces an initial stimulation followed by a depression of the cells of the autonomic ganglia, the central nervous system and voluntary muscle. Its central action may produce convulsions, vomiting and hyperventilation later followed by respiratory paralysis. The

alkaloid constricts the coronary vessels resulting in a diminished blood flow despite a slight elevation of blood pressure

Poisoning—Acute nicotine poisoning may result from idiosyncrasy excessive smoking of tobacco or contact with the alkaloid in insect sprays Two or three drops of pure nicotine are sufficient to cause death before treatment can be instituted If the patient survives after a few moments only restorative therapy is required

Tobacco—Tobacco may be chewed inhaled as snuff or smoked The senior author has no understanding of the pleasures of tobacco chewing From what he has observed of *tobacco chewers* there can be very little absorption of noxious substances since there is undoubtedly a great deal more spitting than swallowing

The use of *snuff* is also difficult of understanding That it must have some virtue is suggested by the universal ownership of elaborate and artistic snuff boxes during the Eras of Elegance Some pleasurable sensation may be produced by the production of a sneeze for purposes of nasal cleansing Certainly not much nicotine can be absorbed and the habit can have little medical significance

The smoking of tobacco is carried out through cigarettes cigars and pipes Lay opinion to the contrary the greatest absorption arises from pipe smoking The cigarette which is the most often condemned is least offensive from the standpoint of clinical practice There seems very little difference in composition between the ordinary smoking products and those which claim to be *denicotinized* The practitioner is warned that the substitution of one for the other will not suffice if smoking is to be interdicted

Attempts to detoxify smoking have been made through the introduction of *holders* many of which contain adsorbent materials such as crystals of one type or another or the length of a dry cigarette Once again it is unlikely that these subterfuges accomplish much by way of detoxification If the physician is certain that clinical symptoms are being produced by smoking the only effectual method of alleviating the disturbance is a complete ban on smoking in all forms and by all devices

Smoking—The use of tobacco is now almost universal Until 25 years ago smoking was a prerogative of the male sex, since then the male consumption of cigarettes is exceeded by women smokers although cigar and pipe smoking remain masculine practices

It is exceedingly difficult to evaluate the effects of smoking since the smoke contains many products other than tobacco The additional ingredients used in the preparation of two popular cigarette brands are as follows

Cigarette 1

Glycerin
Cocoa—22% butter fat
Chocolate liquor—52% butter fat
Maple sugar
Licorice
Peru Balsam
Tolu Balsam
Sweet gum styrax
Jamaica Rum
Cardamom
Coriander
Tonka
Mace

Cigarette 2

Glycerin
Cocoa—22% butter fat
Chocolate liquor—52% butter fat
Maple Sugar
Licorice
Peru Balsam
Tolu Balsam
Sweet gum styrax
Jamaica Rum
Cardamom
Coriander
Tonka
Mace
Deer to me (Apple)

The Effects of Smoking—Besides nicotine the recognized products of smoking include *pyridine bases*, *glycerol*, *furfural* and *acrolein*. The problems arising from smoking thus transcend the pharmacology of nicotine of which 6-8 mg are said to be obtained from a single cigarette.

TABLE 230—CLINICAL CONDITIONS INFLUENCED BY SMOKING

| | |
|------------------------------|---|
| Growth | No evidence that smokers stunt growth |
| Asthma | Clinical evidence suggests that asthma in patients who are underweight may be due to excessive smoking |
| Anorexia | Clinical evidence indicates that underweight smokers gain weight when tobacco is interdicted |
| Hypertension | Make clinical trial before and after test period of 3 weeks of abstinence fairly significant difference—retest after 3 weeks of smoking |
| Tachycardia | Test as with hypertension—significant decrease frequent |
| Cardiac Arrhythmias | As Hypertension (p 200) |
| Angina Pectoris | Test as above—Symptoms change slightly if any |
| Coronary Occlusion | Smoking should be banned |
| Thromboangitis Obliterans | Smoking absolutely forbidden |
| Peptic Ulcer | Test as with hypertension in peptic activity is increased despite negative evidence forbidding during active period and permit while symptoms abate |
| Generalized Arteriosclerosis | Prohibition not warranted unless therapeutic trial is significant |
| Cardiac Failure | Prohibit during decompensation but not after unless therapeutic trial is convincing |
| Pulmonary Tuberculosis | Prohibition unnecessary |
| Emphysema | Prohibition unnecessary |
| Amblyopia | Strict prohibition |

The demonstrable effects of smoking include inhibition of hunger contractions in the stomach increased motor activity in the colon peripheral vasoconstriction transitory elevation of blood pressure and evidences of myocardial irritation as manifested by paroxysms of tachycardia prema-

ture contraction and occasional cardiac pain. The pleasurable effects of smoking are said to be a relief of nervous tension.

As with all other types of drugs, there are marked individual variation. Undoubtedly the toxic evidences noted above are rarely observed in the average smoker. More often they are manifestations of idiosyncrasy or excessive dosage. In consequence of this few of the generalities that are so glibly enunciated necessarily apply to any individual patient. In the end it is necessary to perform a therapeutic test for specific information. This is most easily conducted by prohibition of smoking for a minimum of three weeks followed by a resumption. Those symptoms which disappear during the abstinence period and reappear when the habit is resumed may be stated fairly to be manifestations of nicotineism.

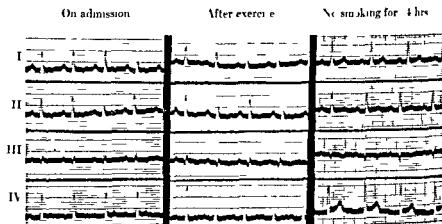


Fig 1127A—M C m 31 Asymptomatic hypertension (150/90) discovered during separation examination. Smoking since the age of 15 averaging 20 packs a day. Smoked 2-4 packs of cigarettes a day while in service.

CHAPTER 181

PHARMACOTHERAPY DRUGS ACTING ON MUSCLE THE URINARY, REPRODUCTIVE AND HEMATOPOIETIC SYSTEMS AND ON INVADING ORGANISMS

Drugs Acting on Muscle

Drugs Acting on Cardiac Muscle

Drugs Acting on Striated Muscle

Stimulants of Smooth Muscle

Depressants of Smooth Muscle

Drugs with Principal Effects on the Urinary Reproductive and Hematopoietic System

Drugs and Antibiotics with Principal Systemic Effects on Invading Organisms

The present chapter deals with a diverse group of drugs whose principal effects are reflected by cardiac visceral or skeletal muscle and those whose chief activities are referable to the urinary reproductive or hematopoietic systems. The section on pharmacotherapy is concluded with a consideration of drugs and antibiotics administered to the human host for the purpose of destroying hostile invading organisms.

DRUGS ACTING ON MUSCLE

Many diverse pharmacologic substances exert various manifestations of activity on muscle. Some exhibit their effects on cardiac muscle while other preparations stimulate or depress skeletal or visceral muscle. In almost every instance the response is amazingly specific considering the similarity of the histologic tissues. *Digitalis* despite its effect on the myocardium is relatively inert so far as striated and visceral muscle fibers are concerned; the *nitrites* have little effect on heart or skeletal muscle yet they violently depress visceral muscle; *curare* responses are limited to skeletal muscle with little appreciable effect on myocardial or visceral muscle.

DRUGS ACTING ON CARDIAC MUSCLE

There are few fields in pharmacology in which there is as much misinformation as that regarding the effects of drugs on cardiac muscle. The truth of the matter is that the only significant stimulant of the myocardium is *digitalis* when administered under certain well defined physiological conditions as detailed with the material on the Circulatory System (p. 770). The activities of heart muscle may be favored by other drugs which do not necessarily exert a direct stimulant effect. Thus *quinidine* aids in the recovery of the myocardium by the restoration of normal rhythm in the cardiac irregularities (p. 802). *papaverine*, *nitroglycerin* and certain of the *xanthines* exert beneficial effects by relaxation of the coronary musculature and secondary augmentation of blood flow. The majority of the alleged cardiac stimulants however exhibit neither primary nor secondary effects which are of benefit to the normal or the failing heart. The

ture contraction and occasional cardiac pain. The pleasurable effect of smoking, are said to be a relief of nervous tension.

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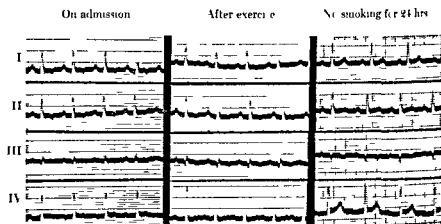


Fig 1127A—M C m 31 Asymptomatic hypertension (150/95) discovered during separation examination. Smoking since the age of 15 averaging 2 packs a day. Smoked 2-4 packs of cigarettes a day while in service.

by curare an alkaloid derived from the stem bark roots and leaves of *Strychnos* plants

Preparations—Until comparatively recently curare was not available for clinical use because of its scarcity and the wide variations in potency of the available drug During the last few years a concentrated aqueous extract of curare (*intocostin*) has been prepared it is assayed by determining the amount required to produce paralysis of the neck muscles as shown by head drop in the rabbit It is marketed in ampoules of which 1 cc = 20 mg

Dosage—The intravenous injection of 10 mg of *intocostin* curarizes 15-20 pounds of body weight males require more than females and older patients more than young ones About 50 to 80 mg are required for a 100 to 150 pound woman and 80 to 120 mg for a 150 to 200 pound man The dose is influenced by the speed of injection small doses given at frequent intervals being more effective than large doses given infrequently

Absorption and Excretion—The actions of curare are elicited only after parenteral administration Following *intravenous injection* the effects are noted almost immediately and reach their peak in two minutes after *intramuscular injection* it requires fifteen minutes for the effects to become apparent

The drug does not act after *oral administration* because absorption is so slow that an effective concentration in the body cannot be established Curare is rapidly excreted by the kidney and a small amount is destroyed by the liver The toxicity of the drug is increased by renal insufficiency

Pharmacology—The principal action of curare is on the skeletal muscles and autonomic ganglia which it prevents from responding to nerve impulses or to acetylcholine It blocks only the nicotinic actions of acetylcholine and does not affect the action of smooth muscle and gland cells (muscarinic actions) The inhibited structures are not paralyzed and are capable of responding to electrical stimulation The chief effect of curarization is a peripheral motor flaccid paralysis which selectively affects first the firm fast moving muscles of high chronaxie (extra ocular muscles and muscles of the face) and later the muscles of the head neck extremities intercostals and diaphragm

The drug also stimulates the central nervous system but this action is masked by the peripheral muscular depression which inhibits the development of convulsions The sensory nerves are not affected and the reflexes though diminished are not abolished

The parenteral administration of curare to man is followed by a characteristic train of symptoms which resemble those of *myasthenia gravis* (p 2886) The first complaint is blurring or haziness of vision This is followed by bilateral ptosis relaxation of the muscles of facial expression tightness of the throat and huskiness of the voice Next there is inability to raise the head and weakness of the muscles of the spine and extremities Lastly respiration becomes shallow due to paralysis of the intercostals and diaphragm The action of the drug is quickly counteracted by an injection of neostigmine (p 3874)

A large number of drugs have a curare like action on skeletal muscle These include quinine erythroidin hydrochloride ammonium bases muscarine and snake venoms

digitalis like response of *calcium* has more theoretic than practical significance and *caffeine* *strychnine* *camphor* *metrazol* and *nikethamide* have no significant effects on cardiac muscle in therapeutic doses despite many extravagant claims to the contrary

Barium potassium chloroform and *chloral* may be highly toxic to the heart muscle. The first produces violent stimulation and the last three show depressant effects

TABLE 21 -- DRUGS WITH PRINCIPAL EFFECTS ON MUSCLE

| | |
|---|--|
| <i>Digitalis</i> (p. 833) | Stimulates cardiac muscle |
| <i>Calcium</i> (p. 603) | Digitalis like effect on cardiac muscle |
| <i>Caffeine</i> (p. 391) | Inconsequential effect on cardiac muscle in therapeutic doses |
| <i>Camphor</i> <i>Metrazol</i> and <i>Nikethamide</i> (p. 3970) | No significant effect on cardiac muscle in therapeutic doses |
| <i>Ephedrine</i> and <i>alldiuretics</i> (p. 3976) | No significant effect on cardiac muscle in therapeutic doses |
| <i>Strychnine</i> (p. 3444) | No effect on cardiac muscle in therapeutic doses |
| <i>Barium</i> (p. 830) | Toxic stimulation of cardiac and smooth muscle may be tried in heart block (p. 830) |
| <i>Quinidine</i> (p. 851) | Depressant action on cardiac muscle useful in treatment of cardiac irregularities (p. 873) |
| <i>Instagramin</i> (p. 39) | Highly toxic to heart muscle |
| <i>Chloroform</i> (p. 3944) | Toxic to heart muscle |
| <i>Chloral</i> (p. 3937) | Toxic to heart muscle |
| <i>Aminoacetic Acid</i> (p. 3448) | Stimulant of skeletal muscle beneficial effects greatly overestimated |
| <i>Physostigmine</i> (p. 3874) | Specific stimulant effect in myasthenia gravis (p. 387) antidote for curarization stimulant of visceral muscle |
| <i>Curare</i> (p. 3888) | Depressant of skeletal muscle |
| <i>Frythrodin</i> (p. 390) | Depressant of skeletal muscle |
| <i>Ooster or Obe</i> <i>lituitary Extract</i> (p. 1178) | Stimulant of visceral muscle especially gut and uterus |
| <i>Histamine</i> (p. 3990) | Visceral muscle stimulant |
| <i>Nitrites</i> (p. 389) | Visceral muscle depressant |
| <i>Nanthines</i> (p. 3866) | Amnophylline intravenously of some value as an antispasmodic of visceral muscle |
| <i>Papaverine</i> (p. 394) | Visceral muscle depressant |
| <i>Demerol</i> (p. 3833) | Visceral muscle depressant |
| <i>Thiocyanate</i> (p. 910) | Visceral muscle depressant in hypertension |
| <i>Garlic</i> | Claims in hypertension unfounded |
| <i>Watermelon Seed</i> | Claims in hypertension unfounded |
| <i>Larsley</i> | Claims in hypertension unfounded |

DRUGS ACTING ON STRIATED MUSCLE

Striated muscle is quite resistant to drug therapy. *Physostigmine* is effective in restoring normal conditions in myasthenia gravis (p. 2886) but it does not strengthen normal structures. Equally disappointing in this latter respect have been the results of the administration of *Aminoacetic Acid U.S.P.* (*Clycocoli*) enthusiastically advocated as a muscle tonic.

Muscle relaxation has recently yielded to pharmacologic endeavors. Depressant effects have been obtained with *intocostrin* a variety of *curare*.

Curare—The response of skeletal muscle to somatic nerve stimulation is mediated through *acetylcholine*. The action of this substance is blocked

action and by bacteria. Histamine used as a therapeutic agent is prepared synthetically from diamino acetone.

Histamine occurs naturally in a wide variety of plant and animal sources chiefly ergot, soybean, rattlesnake venom, intestinal mucosa, muscle, lung, liver, heart and spleen. Appreciable amounts of histamine are present in leukocytes, particularly the eosinophils.

Preparations—The commonly used preparation is histamine acid phosphate which consists of freely soluble colorless crystals. The official solution of histamine phosphate contains 1 mg. per cc. Solutions of the salt may be sterilized in boiling water for thirty minutes without deterioration. In recent years there has been a tendency to express dosage in terms of the free base. The ratio of salt to base is roughly 3:1. Histamine acid phosphate is available in ampoules containing the drug in physiological saline (1:1000 and 1:10,000) and in tablet form. It is also prepared in ointments for the local application of the drug by injection.

Absorption, Fate, Excretion—Histamine is rapidly absorbed from all routes of administration but is only active when given parenterally. Most of the material is rapidly destroyed in the body by deamination and subsequent oxidation and by the action of the enzyme *histaminase*. One unit of histaminase inactivates 1 mg. of histamine in twenty-four hours. The enzyme is a protein, heat-labile and very slow acting. As yet, it is available for parenteral and oral use. It has been recommended in the treatment of conditions ascribed to the liberation of histamine such as *urticaria*, histamine headache and vasomotor rhinitis, but its value is extremely doubtful.

General Pharmacology—The pharmacological activity of histamine is intense and widespread, involving most of the body tissues in some way. The principal actions of the drug are manifest on the cardiovascular system, smooth muscle and the gastric glands.

CARDIOVASCULAR ACTION—Histamine dilates all capillaries and increases their permeability. Plasma and its protein escape into the tissue spaces from the vascular compartments and *localized edemas* are produced. Intradermal injection of histamine phosphate produces a wheal surrounded by a red flare, an effect similar to that resulting from simple types of skin injury in which the H substance believed to be the active agent in inflammation is liberated. The significance of histamine in the production of shock is elsewhere discussed (p. 928).

In man, histamine produces a direct dilatation of the arterioles. The skin temperature rises and flushing is noted. However, the drug is so rapidly destroyed that fairly large doses (10 mg. per hour) may be given intravenously without causing a fall in blood pressure. There is no pronounced direct cardiac action.

SMOOTH MUSCLE—Histamine is a powerful constrictor of the smooth muscle of the bronchioles and causes intense bronchospasm. The bronchoconstrictor action is minimal even with large doses in normal humans, but it is relatively easy to elicit in patients with bronchial asthma (p. 2101). Uterine muscle *in vitro* and *in vivo* is stimulated by histamine.

GASTRIC GLANDS—Histamine has a very powerful stimulating action on the gastric glands and causes the secretion of copious gastric juice. The acid-forming cells, particularly, are stimulated and the juice is very

Therapeutics—Curare has proved of considerable value in the prevention of fractures of the spine and long bones and of visceral trauma among psychiatric patients receiving *convulsive shock therapy* by metrazol or electricity (p 1329) Preliminary curarization is widely practiced in this group of patients The chief hazard is respiratory embarrassment which can be effectively managed by artificial respiration or injections of neostigmine

The intramuscular injection of curare has proved a valuable adjuvant to other methods in the treatment of *cerebral palsy* (p 2948) Progress under physiotherapy is accelerated by doses of from 0.9 to 3.3 mg per kilogram of body weight which produce muscle relaxation lasting several days Curare increases the degree of muscle relaxation during *general anesthesia* and assists in the reduction of *fractures* and *dislocations* It may be useful also in the treatment of *tetanus* and other convulsive disturbances It may relax the painful muscle spasms in the early phases of poliomyelitis (p 457)

Erythroidin Hydrochloride—Erythroidin hydrochloride is an alkaloid with a curare like action It has been used for the same purpose as intocostin but apparently is not as effectual

Pitcher Plant and Ammonium Sulfate—Solutions of pitcher plant have been found of value when administered by local infiltration to areas in which there has been neuralgic pain Identical results may be effected by the simpler method of employing 0.5 to 1 per cent ammonium chloride or 5 cc of distilled water containing 200 to 400 mg of ammonium sulfate

The ammonium salt injections may be given by perineural infiltration paravertebral or intraspinal injection Other than immediate nausea and vomiting in some of the patients and temporary motor effects no untoward reactions have been observed and excellent relief of pain has been reported

STIMULANTS OF SMOOTH MUSCLE

Smooth muscle is most effectively stimulated by the cholinergins particularly physostigmine and neostigmine It also contracts vigorously as the result of the administration of posterior pituitary extract Barium is a powerful smooth muscle stimulant but thus far its effects present no therapeutic possibilities Stimulation of smooth muscle is also a property of histamine despite the fact that the drug produces a fall in blood pressure due to the more overwhelming capillary effects

Histamine—Histamine a substance endowed with intense pharmacological activity is present in most animal tissues and is believed to be liberated as a result of tissue injury Although few substances have greater academic importance the clinical usefulness of histamine is limited Because of its powerful actions on the small blood vessels histamine has been regarded as the chemical agent responsible for the vascular changes occurring during inflammation (p 16) and for the shock (p 928) accompanying the many types of tissue damage The various phenomena of anaphylaxis (p 547) have been ascribed by some to the release of histamine

Chemical Nature and Sources—Histamine is derived from the amino acid *histidine* by removal of the carboxyl group (COOH) This change readily occurs in the body during the splitting of proteins by enzyme

nitrite is the least expensive and most frequently used inorganic preparation

Amyl Nitrite USP is a clear volatile fluid with a distinct fruity odor. It comes in fragile glass pearls, each containing 0.2 cc. It is inhaled as the pearl is crushed and the drug volatilized.

Glyceryl Trinitrate USP known also as *nitroglycerin* and *glonoin* is an explosive yellowish fluid. It may be dispensed in tablets containing 0.6 mg ($\frac{1}{100}$ grain). These tablets are absorbed from the sublingual space.

Erythrol Tetranitrate USP is available in undiluted form in tablets containing 15 to 30 mg ($\frac{1}{4}$ to $\frac{1}{2}$ grain). It is also dispensed with equal parts of lactose (30 to 60 mg).

Mannitol Hexanitrate is sold in tablets containing 15 to 30 mg ($\frac{1}{4}$ to $\frac{1}{2}$ grain).

The fluid preparations of the nitrite are unstable and explosive. Their use is not recommended. There is a 1 per cent alcoholic Spirit of Nitroglycerin and a 4 per cent strength of ethyl nitrite known as Spirit of Nitrous Ether or Sweet Spirits of Niter. In olden times these preparations were used as antipyretics (p. 3832).

Pharmacology—The principal action of the nitrite ion is a depression of smooth muscle which leads to relaxation. The effect is most marked on the capillaries, venules and small arterioles. Here the vasodilatation is accompanied by an increase in local blood flow, an effect which is of therapeutic value in the coronary system but which leads to unpleasant and at times dangerous increases in the tension of the fluid within rigid cavities such as the cranium and orbit. Vasodilatation of the meningeal arteries produces increased intracranial pressure and results in the production of the bursting headache that so frequently accompanies the nitrite effect. Increase in intra-orbital tension may precipitate a glaucoma in susceptible individuals. The smooth muscle depression of nonvascular structures is of lesser clinical importance and usually is not noted unless the fibers are in a state of intense hypertonicity. Thus the nitrite effect on bronchi, the biliary, gastro-intestinal and genito-urinary smooth musculature is only manifest during spasm or colic.

Vasodilatation leads to secondary vascular manifestations. Systemic blood pressure falls and there is a compensatory tachycardia. Systolic pressure is affected more than diastolic. A more pronounced effect is noted when vascular tonus is increased as in hypertension. A lesser effect is noted when the vessels are thickened as in arteriosclerosis.

The increase in the vascularity of the coronary bed favors myocardial function since the heart is enabled to work with greater efficiency.

Therapeutics—The important therapeutic actions of the nitrites are manifested in the treatment of angina pectoris and hypertension.

ANGINA PECTORIS—The nitrites find their principal usefulness in the prevention and relief of the acute paroxysms of cardiac pain that are best described as angina pectoris. The rapidly acting amyl nitrite or nitroglycerin tablets relax the coronary vascular tree and increase blood flow. Systemic blood pressure falls and the pulse rate increases.

acid The pepsin content is also increased The action on the gastric glands is the basis for the use of histamine in testing for *achlorhydria* (p 1681)

Therapeutic Uses GASTRIC ANALYSIS—Since histamine is the most powerful secretagogue for the gastric glands it is used to demonstrate their physiological condition when *achlorhydria* is suspected The increase in HCl production usually begins within fifteen minutes after injection and reaches its maximum in thirty to sixty minutes Anacidity after the subcutaneous injection of histamine base 0.25 to 0.5 mg is a true anacidity In patients with bronchial asthma bronchitis emphysema and cardiac failure histamine may precipitate an episode of difficult breathing even with the small dose used in the test Epinephrine should be readily available in the event that an untoward reaction occurs

HISTAMINE HEADACHE—Histamine headache is characterized by the sudden onset of a unilateral headache of short duration usually in middle aged patients The pain is severe and constant and tends to involve the temple eye neck and face Attacks can be induced in sufferers by injections of small amounts of histamine After histamine is used to diagnose this condition it is given in gradually increasing amounts to accomplish desensitization

ANTI HISTAMINES—The introduction of potent anti histamine preparations promises to revolutionize the symptomatic treatment of allergic and allied conditions Pyribenzamine and benadryl are elsewhere discussed in detail (p 565)

Toxicity—An overdose of histamine leads to an alarming group of phenomena that may culminate in profound shock There is a precipitous fall in blood pressure bronchial spasm dyspnea headache diarrhea and eventual collapse Epinephrine is the *specific antagonist* of histamine and should be given promptly Administration of 15 mg of epinephrine will counteract the effects of 15 mg of histamine

DEPRESSANTS OF SMOOTH MUSCLE

Smooth muscle is most powerfully relaxed by the drugs which act through the involuntary nervous system both the *adrenergics* (epinephrine) and the *belladonna* group of drugs release spastic muscle the former through a stimulation of the depressor nerve and the latter by depression of the motor filaments

As a direct muscle action relaxation is produced by the *nitrites* *papaverine* and *demerol* So far as our experiences are concerned the effects attributed to the *thiocyanates* *garlic* *parsley* and *watermelon seed* are inappreciable if therapeutic doses are used under ordinary clinical conditions

The Nitrites—A number of derivatives of nitrous and nitric acid liberate in the body fluids a nitrite ion that produces prompt relaxation of smooth muscle irrespective of innervation

Preparations—*Sodium Nitrite U.S.P.* is a white crystallized powder freely soluble in water forming unstable solutions It is prepared in tablets of 30 to 60 mg (0.5 to 1 grain) for oral use Sodium

BILIARY RENAL AND INTESTINAL COLIC—On very rare occasions the patient with biliary renal or intestinal colic may be relieved by the administration of the rapidly acting nitrites. Usually the result is disappointing although in laboratory animals a demonstrable fall in intrabiliary pressure regularly occurs.

HYPERTENSION—The use of the nitrites in the treatment of sustained hypertension is unphysiological and disappointing. It is to be recalled that hypertension is commonly the result of a more fundamental cardiovascular or renal disturbance. The drugs which cause a rapid lowering of systemic blood pressure may be both injurious and illogical.

Despite these physiological considerations the various nitrites are widely employed in the management of hypertension particularly of the essential variety. The various members of the nitrite group differ chiefly in their rate of action. The rapidly acting preparations so useful in the treatment of angina are of least value in the management of a sustained elevation of blood pressure while the longer acting preparations are to be preferred.

Still another suggestion to utilize the sustained nitrite action has been presented through the administration of *bismuth subnitrate* in doses of 0.65 gm (10 grains) three times daily. It was thought that the continuous supply of nitrite occurring from the intestinal action on the nitrate would provide for a constant supply of the drug. Unfortunately the method is to be recommended more for its theoretical ingenuity than its practical accomplishments.

Effective lowering of blood pressure by the nitrites is very prone to produce untoward side effects particularly pounding headache weakness faintness and even syncope. Excessive doses occasionally produce met hemoglobinemia.

OTHER USES FOR THE NITRITES—The nitrites are used to lower temperature as anaphrodisiacs and in the treatment of toxic amblyopia.

The antipyretic effect of the nitrites has been suggested through the cutaneous vasodilatation that occurs. Before the introduction of the currently popular analgesic antipyretics (p 3832) Sweet Spirit of Niter was believed to be an effective drug for lowering temperature.

Potassium Nitrate (saltpeter) is widely used as an anaphrodisiac. Presumably the rationale for the action is the supposed prevention of engorgement of the genitalia. There is no excuse for its continued use.

In certain instances of toxic amblyopia with spasm of the retinal vessels due to substances such as tobacco the administration of nitrites may be beneficial.

Toxicology—The most serious side effects of the nitrites result from excessive lowering of the blood pressure while the patient is standing. Faintness and syncope may result and the patient may be injured in the fall.

Less dangerous but more frequent is the occurrence of the headache which presumably results from engorgement of the meningeal vessels and resultant increase in intracranial tension. On much rarer occasions met hemoglobinemia results as an idiosyncrasy.

The use of nitrites in the treatment of cyanide poisoning is discussed elsewhere (p 748).

The amyl nitrite is taken by inhalation while the patient is recumbent. An effectual fall in blood pressure may be attended by a throbbing headache, nausea, vomiting, syncope and even collapse. Nitroglycerin tablets are best introduced under the tongue. Since they cause a less tempestuous fall in blood pressure they are less likely to produce the unpleasant side effects.

The rapidly acting nitrites are employed in the relief and prevention of angina. In the former instance the patient utilizes the drug when necessary to allay discomfort. There is no doubt but that a certain proportion of the effectiveness of the nitrite is psychotherapeutic since placebo tablets are often as effectual as the drug itself. The prophylactic use of nitrites has even greater psychotherapeutic connotation. Many sufferers from angina become convinced that certain activities are provocative of

TABLE 232.—RATES OF ACTION OF THE NITRITES AND NITRATES

| Drug | Dose | Fall B g wt | Maximal Fall Reached | Maximal Duration | Return to Normal | Extent of Symp- tomatic Fall (mm Hg) |
|---|-------------------------------|----------------|----------------------------|---------------------|---------------------|--|
| Amyl nitrite by inhalation (Wallace and Ruge) | 0.2 cc (3 min) | 1 | 5 minutes | | 7 minutes | 15 |
| Spr glyceryl trinitrate | 0.15-0.2 (2-3 min) | 1 | 5 minutes | 2 minutes | 1/2 h | 25-30 |
| Sodium nitrite | 0.15-0.2 gm (2-3 grains) | 5 | 15 minutes | 20-30 minutes | 1-2 h | 32 |
| Erythrol tetranitrate | 0.03-0.06 gm (1/2-1 grain) | 5 | 22 minutes | 2 hours | 5-6 hours | 35 |
| Mannitol nitrate | 0.06 gm (1 grain) | 15 | 2 1/2-3 h | 3 hours | 6 hours | 35 |

An ingenious attempt to utilize the differences in time relationship is effected by the prescription undernoted:

| | |
|------------------------|--------------------|
| ℞ Sodium Nitrite | 0.032 gm (gr 1/2) |
| Erythryls Tetranitrate | 0.008 gm (gr 1/32) |
| Mannitol Nitrate | 0.016 gm (gr 1/4) |
| Ammonium Hippurate | 0.065 gm (gr 1) |

an attack. Fortified by the use of the nitrite they may often be able to carry on without painful stimulus.

A certain tolerance results from the prolonged and repeated use of the nitrites in the management of angina. It has not been my experience that this habituation is as marked as most descriptions would lead the reader to believe. Many patients with angina continue to use the drug effectively over the course of many years. However the factor of tolerance must be recognized and the patient should be urged to use the smallest effective dose and increase the amount as tolerance develops.

BRONCHIAL ASTHMA—The use of nitrites in the treatment of bronchial asthma has been superseded by other depressants of smooth muscle which are free from blood pressure effects. However in an obstinate bronchospasm rapid relief is occasionally afforded by the administration of nitrites when other measures fail.

The asthma powders and cigarettes widely used by the laity rely on potassium nitrate for what virtue they possess.

fortunately fallen into disuse whereas in former days it was widely employed in the treatment of fevers and eclampsia

DRUGS WITH PRINCIPAL EFFECTS ON THE URINARY REPRODUCTIVE AND HEMATOPOIETIC SYSTEMS

The pharmacology of the urinary reproductive and hematopoietic systems is discussed in the sections which deal with their clinical abnormalities

- See The Urinary System (p 227)
 The Female Reproductive System (p 247)
 The Male Reproductive System (p 293)
 The Blood and Blood Forming Organs (p 1035)

The principal pharmacologic effects that may be utilized therapeutically are tabulated in Table 233

TABLE 233—DRUGS WITH PRINCIPAL EFFECTS ON THE URINARY REPRODUCTIVE AND HEMATOPOIETIC SYSTEMS

| | |
|--|---|
| Urinary Antiseptics (p 2256) | Volatile oil lyso methenamine bicarbonate of soda maleic acid sulfonamide streptomycin penicillin |
| Diuretics (p 2257) | Water salts dextrose urea xanthines posterior pituitary extract parathyroid and thyroid extracts mercurials |
| Antidiuretics (p 1178) | Posterior pituitary extract |
| Hormones Acting on Reproductive Organs (p 1149) | Androgen estrogens progesterin adrenal cortical hormone anterior pituitary extracts placental extracts |
| Abortifacients Ecbolics and Oxytocics (p 2311) | Ergot and ergot alkaloids posterior pituitary extract castor oil quinine |
| Uterine Sedatives Antispasmodics and Emmenagogues (p 2512) | Belladonna analgesic antipyretics alcohol ergot progesterin atropine papaverine demerol |
| Aphrodisiacs and Anaphrodisiacs (p 1049) | Androgen estrogen potassium nitrate |
| Spermatoxides | Contraceptives |
| Hematinics (p 1049) | Iron copper manganese liver extract anti-anemic principle folic acid transfusion |
| Hemolysins (p 1046) | Phenylhydrazine radioactive phosphorus irradiation arsenic |
| Leukocyte Stimulants (p 1047) | Nucleoproteins liver extract adenine sulfate pentanucleotide |
| Leukocidines (p 1047) | Radium antix ray P ₃ nitrogen mustards arsenic |
| Coagulants (p 1046) | Whole blood snake venom calcium cevitic acid menadione (Vitamin K) tissue extracts |
| Anticoagulants (p 1050) | Heparin dicoumarin hirudin lephalin |

DRUGS AND ANTIBIOTICS WITH PRINCIPAL SYSTEMIC EFFECTS ON INVADING ORGANISMS

Medical historians of the future may refer to the first half of the twentieth century as the Golden Age of Chemotherapy Since Ehrlich introduced *606* in 1914 for the treatment of syphilis these relatively few years have recorded the perfection of *arsenotherapy* in spirochetal diseases the introduction of *atabrine* and *plasmochin* in malaria the synthesis of *quinine* and the miracles wrought by the *sulfonamides* *penicillin* *streptomycin* and other agents

The specificity of the chemotherapeutic and the antibiotic agents is such that the uses of each are best described with the clinical manifestations of

The Thiocyanates (Sulfocyanides Sulfocyanates and Rhodanates).—The use of the SCN ion has been suggested, because of its similarity to iodide in the treatment of elevation of arterial blood pressure. Potassium thiocyanate has become popular in clinical use for its direct depression of smooth muscle similar to that exhibited by the nitrites.

Potassium thiocyanate is readily absorbed and equally distributed throughout the body fluid a property which has led to its use for the estimation of the volume of interstitial fluid. Excretion is variable. Consequently it is necessary to follow carefully the serum level in order to accomplish a satisfactory concentration and to avoid toxicity.

The Council on Pharmacy and Chemistry of the American Medical Association has advised against the use of the thiocyanates on the ground that there is not sufficient benefit to the patient to justify the risk of toxic manifestations. In consequence there are no official preparations included in the N N R. The available commercial preparations are the 4 per cent *Lixir* and *Tablets* containing 0.065 gm (1 grain) and 0.2 gm (3 grains).

The advocates of thiocyanate therapy believe that the accomplishment of a blood thiocyanate level of 6 to 12 mg per 100 cc is associated with a sustained fall in tension. This level is attained usually by administering 0.3 gm (5 grains) daily for three days. It is maintained by the use of 0.3 to 7 gm (5 to 105 grains) per week according to the patient's excretion rate.

Significant toxicity invariably occurs with blood concentration in excess of 14 mg per cent. The toxic symptoms may be mild such as weakness, fatigue, muscle pains, coryza and toxic eruptions resembling iodism. They may be more severe and include aphasia, confusion, mania, delirium and collapse. In severe poisoning, coma and death occur.

My experiences with the drug have been most disappointing and under no circumstances would seem sufficiently promising to hazard the toxic symptoms that apparently occur in one patient in six.

In an attempt to popularize thiocyanate therapy a test has been devised for blood level estimation. Those physicians who have confidence in the preparation are advised to purchase the testing kit in order to prevent excessive serum concentration of the drug.

Miscellaneous Depressants of Smooth Muscle.—Many other drugs are commonly employed to relax smooth muscle particularly in the management of hypertension. Innocuous and ineffective preparations include derivatives of garlic, parsley and watermelon seed. Additionally many endocrine products are employed including estrogen, androgen, liver extract, pancreatin and kidney, adrenal cortical and tissue extracts. These may have widespread and undesirable side effects.

Potentially dangerous vasodilator substances are *aconite* and its derivatives as well as *veratrum*. These preparations are mentioned only to be condemned.

Aconite contains the alkaloid *aconitine* which depresses the medullary centers producing slowing of the heart rate and a fall in blood pressure. Associated with this central effect is a peripheral action resulting in tingling and a feeling of warmth. Toxic doses of the drug may produce sudden circulatory collapse and death.

Veratrum is usually prescribed as *veratrum viride*. It has an action not unlike aconitine. Potent preparations are dangerous and the drug has

CHAPTER 182

MEDICAL THERAPEUTICS: THE SPECIALIST CONSULTANT

The technic of consultation for diagnostic purposes has received previous consideration (p 3630). Reference to a colleague for consultation to obtain treatment usually presents little difficulty since the patient welcomes the prospect of getting something done. The therapeutic consultation may be required because the practitioner has reached an impasse for the execution of some difficult technical procedure or for the use of some special apparatus. The specialist consultant employs operative and nonoperative forms of treatment. The former usually require hospitalization whereas the latter may be performed in the office or home.

The general indications for specialist consultation in therapeutics are tabulated in Table 235.

Physiotherapist—The range of physiotherapy is indicated in the special section devoted to that subject (p 3784). For the most part the practitioner may obtain the advantages of effective physiotherapeutic measures in his office or in the patient's home.

Physiotherapists vary from the overenthusiastic zealot whose unjustifiable claims disturb the faith of the practitioner to those thoroughly objective and honest specialists in *Physical Medicine* who honor their chosen profession. The latter may be consulted with trust and confidence.

The Anesthetist—The emergence of anesthesia from the days of the ether cone and its tin container has been accompanied by increasing recognition of the value of the specialist anesthetists. These experts in an ever expanding field are of incalculable assistance in both operative and nonoperative procedures.

Operative Anesthesia—In the operating room the specialist anesthetist employs the newer inhalation gases such as *ethylene* (p 4003) and *cyclopropane* (p 4003). These require special equipment and a nicety of judgment in their administration. Smooth prolonged anesthesia is accomplished with little risk and few postoperative difficulties.

Most specialist anesthetists are proficient in *intravenous anesthesia* with sodium pentothal (p 3923) as well as with the more demanding types of local anesthesia by nerve block and epidural and intrathecal administration (p 3922).

Nonoperative Analgesia—In the nonoperative field the specialist anesthetist provides untold relief from intractable pain by alcohol blocks in conditions as dissimilar as *trigeminal neuralgia*, *sphenopalatine neuralgia*, *angina pectoris* and *vasospastic disorders* (p 1394).

The Radiotherapist—Radiotherapy is already so firmly established as a specialty in its own right that many of its exponents have divorced themselves from diagnostic roentgenology. Some specialists employ x ray treatment (p 3796) alone others use radium (p 3797) exclusively.

The radiotherapist has a wide field in the treatment of the *dermatoses* (p 3707) and the *malignancies* (p 572). Because of sensitivity of the

the individual infective processes Table 234 is a mere listing of products. It does not catalogue the local bactericides or the anthelmintics (p 1894)

TABLE 234—DRUGS AND ANTIBIOTICS WITH PRINCIPAL SYSTEMIC EFFECTS ON INVADING ORGANISMS

| | |
|------------------------------------|--|
| Antibiotics (p 88) | Penicillin streptomycin |
| Antimony (p 132) | Potassium antimony tartrate (tartar emetic) USP antimony thio glycollate NNR fuadin (stibophen) NNR neostam solistibosan neostibosan stibamide isethionate |
| Arsenicals (p 116) | Tryparsamide USP acetarsone (stovarsol) NNR arsphenamine USP neoarsphenamine USP mapharsen NNR |
| Atabrine (p 520) | Quinacrine hydrochloride NNR |
| Bismuth (p 128) | Bismuth sodium tartrate (NNR) bismuth and potassium tartrate (NNR) bismuth subsalicylate (NNR) sobisminol mass (NNR) |
| Chaulmoogra Oil (p 277) | Ethyl chaulmoograte USP |
| Emetine (p 529) | Emetine hydrochloride USP |
| Gold (p 2922) | Gold sodium thiosulfate NNR |
| Iodides (p 88) | Potassium iodide USP |
| Mercury (p 100) | Mercuric salicylate (NNR) |
| Oxyquinolines (p 88) | Chinolone USP violone NNR diodoquin NNR |
| Plasmochin (p 520) | Pamaquine naphthoate NNR |
| Quinine (p 520) | Quinine hydrochloride USP totaquine USP |
| Salicylic Acid (p 194) | Sodium salicylate USP |
| Sulfonamides (p 88) | Soluble Sulfanilamide USP sulfathiazole USP sulfa diazine NNR sulfamerazine NNR Insoluble Sulfaguanidine sulfasuxadine sulfaphthalidene Germanin (Bayer 905) moranyl (Fournau) antrypol naphuride (Winthrop) |
| Synthetics (p 533) | Anti malarial (chloroquine) |
| Aralen (p 522) | Anti rickettsial |
| Para aminobenzoic acid (p 136) | |
| p arsenophenylbutyric acid (p 533) | Trypanocide |

CHAPTER 182

MEDICAL THERAPEUTICS THE SPECIALIST CONSULTANT

The technic of consultation for diagnostic purposes has received previous consideration (p 3650) Reference to a colleague for consultation to obtain treatment usually presents little difficulty since the patient welcomes the prospect of getting something done The therapeutic consultation may be required because the practitioner has reached an impasse for the execution of some difficult technical procedure or for the use of some special apparatus The specialist consultant employs operative and nonoperative forms of treatment The former usually require hospitalization whereas the latter may be performed in the office or home

The general indications for specialist consultation in therapeutics are tabulated in Table 235

Physiotherapist—The range of physiotherapy is indicated in the special section devoted to that subject (p 3784) For the most part the practitioner may obtain the advantages of effective physiotherapeutic measures in his office or in the patient's home

Physiotherapists vary from the overenthusiastic zealot whose unjustifiable claims disturb the faith of the practitioner to those thoroughly objective and honest specialists in *Physical Medicine* who honor their chosen profession The latter may be consulted with trust and confidence

The Anesthetist—The emergence of anesthesia from the days of the ether cone and its tin container has been accompanied by increasing recognition of the value of the specialist anesthetists These experts in an ever expanding field are of incalculable assistance in both operative and nonoperative procedures

Operative Anesthesia—In the operating room the specialist anesthetist employs the newer inhalation gases such as *ethylene* (p 4003) and *cyclopropane* (p 4003) These require special equipment and a nicety of judgment in their administration smooth prolonged anesthesia is accomplished with little risk and few postoperative difficulties

Most specialist anesthetists are proficient in *intravenous anesthesia* with sodium pentothal (p 3923) as well as with the more demanding types of local anesthesia by nerve block and epidural and intrathecal administration (p 3922)

Nonoperative Analgesia—In the nonoperative field the specialist anesthetist provides untold relief from intractable pain by alcohol blocks in conditions as dissimilar as trigeminal neuralgia sphenopalatine neuralgia angina pectoris and vasospastic disorders (p 1394)

The Radiotherapist—Radiotherapy is already so firmly established as a specialty in its own right that many of its exponents have divorced themselves from diagnostic roentgenology Some specialists employ x ray treatment (p 3790) alone others use radium (p 3797) exclusively

The radiotherapist has a wide field in the treatment of the *dermatoses* (p 3797) and the *malignancies* (p 572) Because of sensitivity of the

gonadal cells radiotherapy of the ovaries is an effectual method of castration causing fibroid tumors to shrink and precluding the necessity for operative interference

TABLE 235 —THE SPECIALIST CONSULTANT

Otologist (p 2015)

Catheterization and dilatation of Eustachian tubes inflation of middle ear prescription of hearing aid surgery

Rhinolaryngologist (p 2021)

Control epistaxis drainage and lavage of nose and nasal accessory sinuses cocinization of sphenopalatine ganglion insertion of tracheal cannula intubation surgery

Ophthalmologist (p 1540)

Refraction manual expression of lid glands removal of cilia and foreign bodies surgery

Obstetrician and Gynecologist (p 2522)

Fitting of pessary and contraceptive tubal insufflation delivery in suspected dystocia surgery

Dermatologist and Syphilologist (p 3111)

Aid in obstinate dermatoses and treatment resistant syphilis roentgen and radium therapy epilation treatment of neurosyphilis by methods of hyperthermy

Orthopedic Surgeon (p 2807)

Corrective exercises fitting of braces supports corsets and shoes manipulative treatment reduction of fractures and dislocations joint aspiration procaine injections into trigger areas surgery

Neuropsychiatrist (p 1324)

Formal psychotherapy and psychoanalysis convulsion treatments institutionalization

Urologist (p 2244)

Urethral bladder and ureteral instrumentations for dilatation drainage topical applications and irrigations or removal of calculi fulguration of bladder tumors transurethral prostatectomy aspiration of hydrocele surgery

Dentist (p 1663)

Prophylaxis routine dentistry orthodontia fitting of bridges and dentures oral surgery

Physiotherapist

See p 3784

Anesthetist

See p 4002

Radiotherapist

See p 3197

Internist

See p 3901

Pediatrist

See p 2123

Surgeon

See Minor (p 3909) and Major (p 3992) Surgery

Radiotherapy also finds a broad utility in the treatment of *blood dyscrasias* and certain of the *infectious granulomas* the *leulemias* *polycythemia* and *Hodgkin's disease* may be controlled for many years through

expert irradiation. The more recent introduction of radioactive phosphorus (P^{32}) may provide the practitioner with a simple oral method for systemic roentgen therapy.

The internist—The scope of the internist is difficult of definition. Indications for consultation vary with the personal factors relative to the capabilities of practitioner and consultant.

The internist is employed in the treatment of the patient who presents a difficult or refractory problem. He should be employed as a *referee* when there are differences of opinion between practitioner and specialist technician. The internist is acquainted with newer therapeutic procedures such as the administration of recently introduced drugs and biologicals; he may be particularly trained in certain *technical procedures* such as the induction of pneumothorax; he may be more than ordinarily skillful in setting up an intravenous drip for infusion or the administration of sera.

Subspecialization—In the larger medical communities and fully staffed institutions, internal medicine is subdivided into a variety of component parts. Thus there are established departments of *gastroenterology*, *hematology*, *cardiology*, *allergy*, *phthisiology*, *metabolism*, *diagnosis*, *therapy* and *endocrinology*.

The specific therapeutic procedures in which the subspecialists excel are listed below:

Gastroenterologist—For esophageal dilatation (p. 1723), gastric duodenal and intestinal intubation (p. 1749), for aspiration and injection, local procedures by sigmoidoscopy, the treatment of anal lesions, especially hemorrhoids.

Hematologist—For transfusions and infusions.

Cardiologist—Dilatation (p. 854), estimation of operative (p. 5997) and obstetrical risks (p. 2693).

Allergist—For desensitization.

Phthisiologist—Relative to climatotherapy for the induction of pneumothorax (p. 2033) and decisions concerning surgery (p. 2009).

Metabolist—For dietotherapy (p. 658).

Therapist—For the newer drugs.

Endocrinologist—Relative to hormone therapy (p. 1149).

Intensive specialization within internal medicine has certain advantages, but it also has important deterrents. Obviously the man who is especially concerned with a small field is better able to focus his attention upon the subject in which he has a special interest. In many instances, however, the subspecialist cannot see the forest for the trees and loses sight of the broader concepts that are so particularly important for the complete practitioner. No summary of the situation has improved upon the witticism that the specialist is the physician who knows more and more about less and less.

The practitioner often finds himself in the position of having to balance his consultant specialist. Through the practitioner's intimate knowledge of the patient, he is more influenced by the state of general well-being than he is alarmed by a high systolic tension, a cardiac murmur, or an abnormality in the electrocardiographic tracing. He focuses more upon the emotional circumstances involved in peptic ulcer than the amount of acidity in the gastric content. He has little patience with diagnosis as a means to an end and recognizes that this branch of medicine and therapy cannot be

dissociated. He evinces mild amusement when the metabolist measures the patient's intake and output in cubic centimeters and grams, knowing full well what will happen when the patient leaves the metabolism ward. He is more than mildly sceptical of the generalizations enunciated by some endocrinologists.

Pediatricist—Pediatrics is an important subdivision of internal medicine; it varies only in the age of the patient under observation. In most communities, owing to the inadequate supply of pediatricists, the internist takes care of the younger generation as well. He should certainly summon the pediatric consultant, however, for difficult feeding problems, the management of the acute exanthems and other infections peculiar to the early years of life.

Surgeon—See *Minor and Major Surgery* (pp. 3909-3992).

Nonoperative Treatment by the Specialist Consultant—Nonoperative therapeutic procedures performed by the specialist give rise to many difficult problems for the practitioner.

Idherence to Treatment—The practitioner may have to persuade the patient to continue an adequate number of treatments. The patient seeks to stop treatment prematurely for a variety of reasons. Painful procedures such as the washing of an antrum lead the sufferer to conclude that the discomfort of the underlying condition is preferable to that of treatment.

Cessation of Treatment and Overtreatment—Sometimes the practitioner feels impelled to suggest the discontinuance of nonoperative treatment. One can get into the habit of washing a nose or a sinus, of inserting a tampon or ainting a lesion, or of indulging ad infinitum in physiotherapy or mechanotherapy.

Miracle Men—In the course of the treatment of stubborn or obstinate symptoms and complaints the patient usually hears of some local or distant miracle worker who boasts of cures of self-limited diseases and the variable symptomatic disorders such as eczema, rheumatism, bladder weakness, arthritis, female weakness, and the like.

The modalities employed by such miracle men include secret prescriptions, the laying on of hands, injections of imported biological products, and the use of electrical equipment (mostly featured by noise, vibration, crackling lights, and the impressive pulling of switches). After some experience with therapeutic miracles, the practitioner better understands the wisdom of Benjamin Franklin—he is the greatest doctor that knows the largest number of useless remedies.

Negative knowledge unfortunately pays no dividends. Usually, for his mature and honest judgment, the practitioner earns for himself the reputation of being a skeptic and a therapeutic nihilist.

Operative Treatment by the Specialist Consultant—The general practitioner encounters many difficulties in guiding his patient through an operative procedure (p. 3993). The problems that arise involve operative indications and risks, preoperative and postoperative treatment, the management of convalescence and rehabilitation. See *Major Surgery* (p. 4001).

The scope of the preoperative discussion is wide and varied (p. 4000). The practitioner must recognize the indications for operation, he explains the projected procedure to the patient and his friends, he estimates morbidity rate, mortality rate, discomfort, the period of incapacitation, the late

results and the possible postoperative difficulties. At the termination of this recital he must give a definitive judgment as to whether the end justifies the means.

An objective method of arriving at a decision is the projection of oneself into the position of the patient. The practitioner may question whether he would permit himself to be subjected to the proposed procedure for the reasons noted. Would he permit his wife, his child or his parent to be operated upon under similar circumstances?

There is often a wide discrepancy between the indications that specialists set for their patients and those they employ upon themselves and their families. Some have a differential type of indication in that they are apt to be radical in their own field and ultraconservative in the operative specialties of their colleagues. The rhinologist is easily persuaded to have a tonsillectomy on a member of his family or himself but it is not so simple to get him to be cystoscoped or to subject himself to prostatectomy.

Operative Indications—Differences of opinion often arise between practitioner and specialist consultant with regard to operative interference. In general the indications set by the specialist are broader than those of the practitioner since the enthusiastic proponent of a therapeutic endeavor may never evaluate or even have knowledge of extraneous factors and conditions. The rhinologist regards each tonsil as suspect. The dentist intent on a tooth extraction does not pause to find out that the pain in the knee which he hopefully considers as a manifestation of focal infection may result from faulty foot mechanics. Presenting symptoms possibly attributable to the local condition are glibly claimed for the disorder. Parallel cases are cited, statistics are presented. If all else fails the specialist argues that therapeutic test should be applied, he suggests that the tonsils or teeth be removed and the relationship discussed after the postoperative evidence has been culled.

When the patient directly consults the specialist and receives a promise of relief the practitioner has the thankless task of reopening the debate after the problem has been supposedly settled. No matter what may be the final outcome of the anteoperative discussion the practitioner is rarely the gainer. If the operative procedure is permitted and the patient fares well all the credit goes to the specialist; if things go badly the practitioner bears the burden of more than his fair share of the blame. If the operation is deferred or vetoed and the patient continues to do badly it is the practitioner who must bear the brunt of the criticism but should the patient spontaneously recover the specialist gets the credit.

Operative Risks—In the procedures of lesser importance specialists lightly pass over the discussions of operative risk, postoperative discomfort and incapacitation. They are apt to state that the risk is negligible, the discomfort minor and the period of incapacitation virtually nonexistent. The practitioner however recalls many so-called minor procedures (tonsillectomy or hemorrhoidectomy) in which hemorrhage is not infrequent, pain is severe and of long duration and overnight incapacitation has extended to a period of several weeks.

In the *major procedures* some surgeons exaggerate the real risk. It seems to be their viewpoint that the patient and his relatives should be told the worst so that a successful issue will be more appreciated. Other surgeons

underrate the risk by referring to published results ignoring the fact that published mortality is not general mortality that the author of a paper has probably had unusual training and experience and that a large clinic may publish an immediate operative mortality of a fraction of a per cent for a procedure such as a subtotal thyroidectomy in hyperthyroidism while the average surgeon who does the operation only occasionally faces a much higher jeopardy. There is a human element in each technical procedure. Blanket statistics are not necessarily applicable to the individual surgeon and his patient.

The Individual or Patient Risk—The specialist estimation of operative risk deals only with the technical procedure and the practitioner must add to it whatever risks arise from the general systemic status of his individual patient. These include tangible factors (hypertension glycosuria an old tuberculosis or a valvular defect) and the intangibles such as emotional and nervous instability the will to recover or the desire to remain invalided. The practitioner knows from long acquaintance and previous experience which of his patients has the character to withstand adversity and which will be likely to wilt under pressure. In the vernacular he recognizes those who have guts and those who have not.

Not infrequently however the physician's ante operative judgment of the patient's character is upset by actual performance. Thus the highly intense anxious preoperative patient may check his neuroticism at the front door and a surgical experience that promised to be harrowing turns out to be uneventful. On the other hand many braggarts who boast of not having 'a nerve in the body' collapse from trivial traumas such as the pricking of a finger for a blood count.

Ante operative Treatment—With *emergency surgery* (p 3996) there is neither the time nor the opportunity for ante operative treatment. In *procedures of choice* (p 3996) considerable postoperative difficulty may be prevented by intelligent ante operative treatment (p 4000).

The Practitioner in the Operating Room—The practitioner should accompany his patient to the operating room. This discipline serves a number of purposes. Besides the fact that patients derive great comfort from the knowledge that their doctor is at hand the practitioner himself profits when he observes his colleague at work evaluates the specialist's technical skill and operative judgment and follows the steps in the procedure.

During the routine and uneventful operation the presence of the practitioner is a mere gesture. Difficult situations occasionally arise however in which the participation of the practitioner may be of the greatest value. Many surgeons have the furor operandi and must be prevented from doing too much for too long in the face of anesthetic difficulties. It may be the greater part of wisdom to desist for the time being and attempt the procedure at a later date. Under these circumstances it is sometimes difficult to get the surgeon to stop. He is intent upon completion of his task and regards discontinuance of operation as a confession of failure and an offense to his vanity. Tempting the ill humor of the technician the practitioner may call for time to discuss the wisdom of proceeding. The additional seconds consumed in the council add inappreciably to risk and strategic retreat at this time may save life.

With operations that may be done in multiple procedures such as the

rosectomies the risk of completion of procedure at one sitting may be many times the risk of two stage or even three stage operation

Postoperative Report to Relatives—At the completion of the technical procedure the thoughtful practitioner communicates with relatives and friends who in many cases remain in the rooms or halls of the hospital. It is a comfort for them to know that the procedure has been completed and the patient has survived the experience

Postoperative Treatment—The practitioner must not desert his patient after operation. Many specialists resent interference with orders and the practitioner must tread cautiously in his minor and supporting role. An optimum arrangement provides for the care of the wound by the specialist leaving the general orders for the practitioner. Since professional visits are usually made at different times there should be agreement that neither physician will leave any important order without notifying his colleague.

Specialists often have postoperative routines which may include drugs to which the practitioner knows his patient has an idiosyncrasy. Orders for the relief of pain must not be regulated by rule of thumb since discomfort varies in intensity and patients differ in their ability to take punishment. The presence of a postoperative complication calls for a personal consultation between practitioner and specialist. An elevation of temperature may be caused by a local infection in the wound that is the province of the specialist; it may also arise from a pneumonitis that is the realm of the practitioner.

Friendly cooperation between technician and practitioner is of inestimable value in postoperative treatment.

Convalescence and Rehabilitation—The healing of the wound often terminates the relationship between patient and specialist. The practitioner has then the task of dealing with the minor and irritating aftermaths later guiding his patient to the resumption of normal social and economic status (p. 4117).

Recovery from operative surgery is usually punctuated by many and diverse annoyances. There may include a certain amount of lumbago in the back, constipation, paresthesias of the feet, flabbiness of the musculature, surprising weakness in the legs, insomnia and asthenia. These trivia the specialist commonly brushes off with little memory. Oftentimes it is his opinion, either tacit or spoken, that the patient complains unnecessarily and is a neurotic. The unfortunate practitioner finds the problem deposited in his lap and must exercise all of his ingenuity to prevent the patient from becoming discouraged or from seeking treatment in other hands within or without the bounds of the profession.

SECTION XXIV
SURGICAL THERAPEUTICS

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Associate Editors GERSON LESNICK M D
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CHAPTER 183

MINOR SURGERY

- The Armamentarium of Minor Surgery
- The Preparation and Sterilization of Surgical Material
- The Care of Instruments, Supplies and Solutions
- The Preparation of the Surgeon
- The Preparation of the Patient
- Anesthesia
- The Scope of Minor Surgery
- The Planned Minor Surgical Procedure
- The Minor Surgical Aspects of Trauma, Contamination and Infection
- The Management of Wound Complications

Minor surgery is minor only in the sense that it rarely involves serious surgical risk. It is often major in the judgment required, the technic employed and in its economic importance to the patient. It has been estimated that 25 to 60 per cent of the income of general practice is derived directly or indirectly from minor surgery.

In institutional work, minor surgery is performed in the out-patient department or the emergency ward. Usually the services are rendered with a minimum of supervision by the least experienced members of the staff. Meantime major surgical procedures (which the practitioner will rarely if ever be called upon to perform) are being executed in amphitheatres before admiring audiences. The medical student who plans to enter general practice might better occupy the period of internship activity in the performance of the minor procedures.

THE ARMAMENTARIUM OF MINOR SURGERY

The following is a list of the minimum equipment required for performance of the more common minor surgical procedures.

APPARATUS, BANDAGES AND SURGICAL SUPPLIES

- 1 Hand basin with hot and cold running water
- 2 Dispenser for liquid soap preferably green soap
- 3 Stiff nail and hand brush
- 4 Orange sticks
- 5 Fingercots
- 6 Rubber gloves
- 7 Towels and sheets for drap spinal towels
- 8 Gauze flats in sterile packages
- 9 Gauze bandages in 1/4 1 2 3 and 4 inch rolls of 5 yard length
- 10 Gauze and cotton sponges
- 11 Gauze drainage material in tubes (plain and iodoform)
- 12 Strip of rubber tissue for drainage material Use fragments of torn gloves and segments of rubber bands
- 13 Strips of rubber tissue for wadding
- 14 Filter of gauze rolls and plants
- 15 Silicate bandages of the A type
- 16 Adhesive plaster in 1/4 1 2 and 3 inch widths

- 17 Convenient lengths of adhesive plaster with sterile gauze of the Band-Aid type
- 18 Liquid adhesive in tubes
- 19 Colloction in rubber-stoppered bottle with a glass rod applicator
- 20 Safety pins ampoules of sterile procaine (2%) epinephrine 1:1000
- 21 Tongue depressors for finger splints
- 22 Labeled eight-ounce bottles of 7 per cent tincture of iodine 3¼ per cent tincture of iodine 95 per cent alcohol ether benzine green soap 10 per cent formalin for biopsy material
- 23 Electric sterilizer with three speeds and an automatic shut off
- 24 Ordinary pressure cooker for autoclaving dry dressings and gloves
- 25 Vials of penicillin (100 000 units) ampoules of sterile saline for dilution

INSTRUMENTS FOR MINOR SURGERY

- 1 Scalpels preferably with detachable blades of various sizes and shapes
- 2 Blunt pointed and curved scissors
- 3 Small fine sharp pointed scissors
- 4 Bandage scissors
- 5 Probes and grooved directors
- 6 Small artery clamps (6) straight clamps (2) curved clamps (2) large toothed clamps (2) and retracting clamps (2)
- 7 Long dressing forceps to be kept in lysol and used for removing instruments from the sterilizer to the operative field
- 8 Various types of forceps including small smooth and small rat tooth types fine thumb forceps
- 9 Straight and curved needles round and cutting edges
- 10 Needle holders with locks
- 11 Suture material silk C1 and 2 in spools cotton A B and C steel wire #32 and #10 plain catgut 00 and 0 chromic catgut #1 and #2
- 12 Towel clips for anchoring the towels to the skin surrounding the operative field
- 13 Small sharp and blunt retractors as well as small self retaining retractors which are to be used when technical assistance is not available
- 14 Cast cutter cast knife
- 15 Syringes (2 cc 5 cc 10 cc and 20 cc)
- 16 Hypodermic needles assorted 18 g to 26 g and 1½ to 1½ inches long

THE PREPARATION AND STERILIZATION OF SURGICAL MATERIAL

Aseptic surgery demands meticulous care in the preparation of all material in order to prevent contamination of the wound. Bacteria are killed or removed by mechanical cleansing with soap and water by drying by heat and by the use of chemicals. In the large hospital special rooms and assistants are set aside for the preparation of surgical material. This is not practicable in private practice particularly for the physician who has only occasional use for many of the necessities.

It is wise and often economical to take advantage of the facilities of the surgical supply houses which sell sterile materials and solutions. It is possible to purchase at relatively small cost and with great convenience the following sterile material: Gauze flats in packages gauze towels in packages gauze bandages in rolls gauze and cotton sponges in containers packing materials both plain and iodoform in tubes ampoules containing the various brands of the cocaine substitutes for local anesthesia ampoules containing sterile saline solution dextrose and Ringer's solution suture materials silk catgut chromic and horsehair with or without needles. The remaining material must be freshly prepared in the office. This can be accomplished by scrubbing with soap and water sterilization by moist heat sterilization by dry heat and chemical sterilization.

Mechanical Cleansing—Vigorous scrubbing with soap and water is the most important method of decreasing bacterial contamination. Instruments and dressings should not be placed in the sterilizer until thorough washing has been accomplished. This is extremely important when instruments have become contaminated with exudates and wound secretions. *Before and after use* instruments are scrubbed for at least five minutes with green soap and water employing a brush or a swab of cotton or gauze. Then they are dried and replaced in the instrument cabinet.

Sterilization by Boiling—Simple boiling of instruments for ten minutes kills all forms of bacteria. However, spores are resistant to this treatment. Instruments or dressings contaminated with sporulant organisms (tetanus, gas gangrene) require more thorough sterilization such as two autoclavings for 20 minutes at 15 pounds pressure.

Instruments and dressings that have not become grossly contaminated are prepared by preliminary scrubbing with soap and water followed by boiling for ten to fifteen minutes. The electric sterilizer is the most convenient for this purpose. In an emergency materials may be boiled over the gas or wood stove or open fire. The addition of a little washing soda to the water in the sterilizer may prevent rusting and increase the efficiency of the sterilization.

Dry Sterilization—Dry goods are satisfactorily sterilized in an autoclave. For this purpose the relatively inexpensive pressure type of cooker may be employed. The materials for sterilization are neatly and lightly packed in bundles usually made of toweling. The packages are placed in the cooker which is set on the stove. Steam is allowed to escape from the vent of the cooker for at least ten minutes to insure the evacuation of all air from the system.

For complete sterilization pressure is maintained at 15 pounds for twenty minutes. An exception to this is the preparation of rubber goods such as gloves which can withstand only ten to fifteen minutes at this high temperature and pressure.

At the end of the sterilization period the steam is permitted to escape while the sterilizer is still hot. The condensed moisture evaporates and the goods are obtained in a dry state.

Chemical Sterilization—The boiling of sharp instruments results in dulling of the edges. It is better therefore to sterilize scalpels, etc. with a chemical such as cresol lysol or one of the commercial solutions sold for this purpose. Rubber goods particularly silk and rubber catheters deteriorate when boiled. They are kept sterile by immersion in a 1:1000 solution of bichloride of mercury. Exposure of at least an hour is necessary for chemical sterilization.

THE CARE OF INSTRUMENTS SUPPLIES AND SOLUTIONS

Instruments are scrubbed and washed before sterilization. Following use they are scrubbed again. Cutting edges are sharpened on a stone or hone. A small amount of light mineral oil is applied to joints and cutting surfaces.

Blades are wrapped in cotton and stored in a safe and dry place such as an instrument cabinet. Rubber goods such as gloves, catheters and tubes must be completely dried if rotting is to be prevented. They may

be protected by the liberal use of a talcum powder *Glassware* particularly syringes and needles is sterilized by boiling Preliminary to sterilization foreign material such as blood and exudate is mechanically removed from pistons and barrels and from the lumen of the needle

After use *syringes* and *needles* are rinsed first with cold and then with hot water A lubricated stylet is placed in the lumen of each needle Syringes which have contained oily solutions are cleansed first with benzine

Sterile solutions are best purchased in ampoules or flasks of varying sizes When large amounts of fluid must be used for irrigation it is economical to improvise solutions A satisfactory physiological saline solution is prepared by adding a level teaspoonful of table salt to a pint of water The solution is brought to boiling point on three successive days In the intervals between the periods of boiling spores germinate into the living forms of bacteria which are then susceptible to moist heat

THE PREPARATION OF THE SURGEON

Preparatory to the execution of any minor surgical procedure the practitioner carefully scrubs his hands with soap and running water A stiff brush is vigorously employed Particular attention is paid to nails and cuticles The orange stick removes dirt from under the free border of the nails

Gloves are not needed for lesser procedures particularly where instruments can be employed and contact avoided between hand and operative field Instruments are arranged on the tray so that their handles all point one way and do not come in contact with the tips of other instruments After use they must be carefully replaced in the same position

If the procedure is of any magnitude the use of sterile gloves assists in maintaining the aseptic technic of operation For his own sake the practitioner should use rubber gloves in dealing with an infected wound particularly one that is grossly contaminated or which is possibly of syphilitic origin In the absence of rubber gloves finger cots may be employed They are scrubbed as in the preparation of the rest of the hands and fingers Gowns and masks are rarely necessary in the performance of minor surgical procedures of short duration If the surgeon is suffering from a respiratory infection he should use a mask which may be improvised with several layers of gauze

THE PREPARATION OF THE PATIENT

In his enthusiasm for the care of the local condition the practitioner must not lose sight of his responsibility to care for the patient Preliminary to planned surgical procedures (p 3927) *sedative drugs* are used as antidotes to the local anesthetic and to allay apprehension In the more formidable procedures when spinal or general anesthesia is required the patient is entitled to pre-anesthetic medication with a *barbiturate* a *hypnotic* *avertin* or a combination of *scopolamine* with *morphine* *dilaudid* or *demerol*

In the surgery of trauma (p 3950) the patient receives larger doses of pre-anesthetic medication since there is always an element of shock With contamination of a wound it may be a wise precaution to administer alone or in combination *sulfonamide* *penicillin* or *tetanus gas* *gangrene anti toxin* The most vigorous precautions are required in bites

and cat bites suggest a possible rabies (p. 439) snake and spider bites pose the question of specific venom (p. 3970) human bites are often a portal of entry for the organism of syphilis (p. 3970)

ANESTHESIA IN MINOR SURGERY

Minor surgical procedures can be successfully executed without the production of unnecessary pain. The methods of anesthesia have been made readily available so that many can be performed without professional assistance in the office of the practitioner.

TABLE 26—NOTES ON PRE-ANESTHETIC MEDICATION

Phenobarbital 15 mg. ($\frac{1}{4}$ grain)

Preliminary to minor procedure

Secobarbital (Hypnocal) 45 mg. 90 mg. or 180 mg. ($\frac{1}{4}$, $\frac{1}{2}$, or 3 grains)

Preliminary to longer or more painful procedure and before spinal and inhalation anesthesia. Use largest dose before basal anesthesia with or without morphine or demerol and scopolamine. Avoid before pentothal.

Morphine 15 mg. ($\frac{1}{4}$ grain) Dilaudid 2 mg. ($\frac{1}{50}$ grain) or Demerol 50 mg. (1 grain)

By hypodermic if patient is in pain or shock. Preliminary to more formidable procedures.

Scopolamine Hydrobromide 0.5 mg. ($\frac{1}{40}$ grain)

Combine with morphine dilaudid or demerol particularly before general or spinal anesthesia. Use alone before pentothal.

Atropine Sulfate 1 mg. ($\frac{1}{100}$ grain)

Prefer scopolamine

Ephedrine Sulfate 22 or 45 mg. ($\frac{1}{2}$ to $\frac{3}{4}$ grain)

Give orally with phenobarbital or hypnotic before spinal anesthesia to prevent hypotension.

Anesthetize with Amyl Nitrate Hydrate 70 to 80 mg. per kilogram of body weight

Give freshly prepared rectal retention enema in place of hypnotic before inhalation or topical anesthesia. Avoid morphine and demerol.

Sulfadiazine 4 gm. (60 grains)

For contaminated lacerated and puncture wounds

Penicillin (50,000 to 100,000 Oxford units)

For contaminated lacerated and puncture wounds. Also intramuscular

Tetracycline 2 Grams Ampicillin (Combination Syringe Package)

For soil and fecal-contaminated wounds

LOCAL ANESTHESIA

In general one of the methods of local anesthesia can be made available for all minor surgical procedures.

The Advantages and Uses of Local Anesthesia in General Practice.—For the general practitioner local anesthesia is the method of choice. The advantages are summarized below:

1. Local anesthesia requires minimal equipment.
2. There is no need for a surgical assistant since the induction of anesthesia can be accomplished by the practitioner himself.

- 3 The patient *retains consciousness* and may leave the office as soon as the operative procedure has been completed
- 4 In certain operations as in the nose and throat it is important for the patient to *retain the pharyngeal reflex* and prevent the aspiration of blood and infectious material into the lower pulmonary passages

TABLE 237.—PREPARATIONS AND INDICATIONS FOR ANESTHESIA IN MINOR SURGERY

| Drug | Technic | Indication |
|---|------------------------|--|
| Ethyl Chloride | Local freezing | Simple incisions Relief of pain in sprain or contusion |
| Cocaine Substitutes Metycaine Alpin Butyn or Larocaine (1-2 per cent) | Topical application | For mucous membranes and open wounds For removal of foreign bodies cleansing and irrigation For instrumentation of body cavities |
| Larocaine or Diothane (1-2 per cent) | Infiltration | For small clean areas where there is no danger of distorting tissues or propagating infection along tract of needle For sprains, strains, trigger points and fractures |
| Procaine or Diothane (1-2 per cent) | Field block | For larger areas to avoid distortion of anatomy and guard against spread of infection Permits use of retractors |
| Procaine (1-2 per cent) | Nerve block | For fingers toes and hands |
| Procaine or Metycaine (1.5 per cent) | Epidural block | For perineum under institutional conditions |
| Procaine (50 mg) | Spinal | For abdomen perineum and lower extremities under institutional conditions |
| Pentothal Sodium | Intravenous anesthesia | For short procedures if assistant is available |
| Ethyl Chloride, Vinyl Ether and Chloroform | Inhalation anesthesia | For longer procedures preferably in hospital where assistants are available |

The Limitations and Disadvantages of Local Anesthesia —Local anesthesia has the following limitations and disadvantages

- 1 Local anesthesia requires the *cooperation of the patient* In dealing with infants young children and highly sensitive and uncooperative adults the apprehension and struggling of the patient may make it necessary to utilize general anesthesia
- 2 Where several neoplasms or cysts must be removed the entire operative area cannot be included in a single anesthetic field Under such circumstances it may be wiser to produce loss of consciousness

Local Anesthetic Preparations—Local anesthesia may be effected by freezing with *ethyl chloride* or by the use of *cocaine* or one of its substitutes

TABLE 238.—PREPARATIONS AND USES OF COCAINE SUBSTITUTES

| Preparation | Strength | Use |
|---|------------------|----------------------------------|
| Alypin Hydrochloride N.R. | 0.5 per cent | Topical application |
| Butyn Sulfate N.R. | 2 per cent | Topical application ointment |
| Diothane Hydrochloride N.R. | 1 per cent | Injection or topical application |
| Ethyl Aminobenzoate U.S.P. | 0.10 per cent | Powder ointment or lozenge |
| Eucocaine Hydrochloride N.R. | 2-5 per cent | Topical application |
| Metocaine Hydrochloride N.R. | 0.5-1 per cent | Injection |
| Metocaine Hydrochloride N.R. | 2 per cent | Topical application |
| Nupercaine Hydrochloride N.R. | 0.0-0.1 per cent | Injection |
| Nupercaine Hydrochloride N.R. | 0-2 per cent | Topical application ointment |
| Ulenacaine (Holocaine) Hydrochloride N.R. | 1 per cent | Topical application |
| Procaine Hydrochloride N.R. | 0.1-2 per cent | Injection |
| Procaine Hydrochloride N.R. | 1-2 per cent | All purpose |
| Tutocaine Hydrochloride N.R. | 1 per cent | Topical application |

Preparations available in 1 or 2 per cent solution in sterile ampoules as sterile crystals in ampoules for spinal anesthesia as bulk crystals and as tablets for solution in sterile water. Tablets usually combined with epinephrine so that 1 or 2 per cent of local anesthetic has 1:25,000-1:50,000 epinephrine.

TABLE 239.—PHARMACOLOGY OF COCAINE AND ITS SUBSTITUTES

Sensory Nerve Ends

Local application produces selective abolition of sensation of pain but retention of sense of touch.

The local cocaine effect does not reach a peak until several minutes after injection. Incision and instrumentation should be deferred to permit the full effect of the drug.

Adrenergic Nerve Ends

Stimulation causes local vasoconstriction with pallor, ischemic and delayed absorption. Latent action potentiated by epinephrine (1:20,000 to 1:50,000) increases intensity and duration of local anesthesia and lessens local bleeding.

Cerebral Cortex

Stimulation after absorption usually as an idiosyncrasy always toxic prevented and treated by barbiturate.

The undesirable cortical stimulation and adrenergic effects are often seen in patients with hyperthyroidism (p. 1197). If local anesthesia is used in these individuals the preliminary sedation with barbiturates should call for double doses.

The cocaine jag may lead to addiction. Cocaine sprays and office treatments are to be avoided in sensitive and highly neurotic patients who suffer from chronic disturbances such as infection in the paranasal accessory sinuses.

Adrenergic System

Stimulation after absorption. May produce tachycardia, hypertension, mydriasis and mania may be due to epinephrine potentiation. Treat with barbiturates using pentothal sodium intravenously if necessary.

The undesirable cortical stimulation and adrenergic effects are often seen in patients with hyperthyroidism (p. 1197). If local anesthesia is used in these individuals the preliminary sedation with barbiturates should call for double doses.

Ethyl Chloride—Ethyl chloride U.S.P. is a colorless fluid marketed in glass tubes equipped with a nozzle through which the drug is sprayed directly upon the area to be anesthetized. To apply it the tube is inverted.

and the spray directed at the operative site. The container is held at a distance of approximately 12 to 18 inches from the skin area. Shortly frosting occurs at the sprayed site and it is possible to make a small and superficial incision painlessly through the frozen area. This type of local anesthesia is satisfactory only for *simple incisions* since further operative work is not possible without the production of pain.

The use of ethyl chloride spray has been extended to the treatment of *contusions sprains* and the sites of *referred pain*. Through a mechanism not well understood a reflex relief of pain is experienced. With early active motion disability and discomfort are sharply reduced.

Cocaine and Cocaine Hydrochloride—The use for local anesthesia of cocaine and the cocaine substitutes constitutes one of the greatest contributions to medicine. The pure alkaloid of cocaine is never employed because of its insolubility. The alkaloidal salt Cocaine Hydrochloride USP, may be administered to the normal adult in doses approximately 15 mg ($\frac{1}{4}$ grain). A maximum dose is 60 mg (1 grain) or the equivalent of 1.5 cc of a 1 per cent solution.

While the local effect of the alkaloidal salt is highly satisfactory its clinical use is limited through its stimulation of the sympathomimetic system and cerebrum and the danger of habituation or addiction. Because of these undesirable effects various cocaine substitutes have been introduced. These aim to equal the local efficacy and to avoid the constitutional symptoms of the pure alkaloidal salt.

THE TECHNIC OF LOCAL ANESTHESIA

Local anesthesia may be produced by topical application, local infiltration, field block, nerve block, epidural or caudal block and by spinal anesthesia.

Topical Anesthesia

The accessible *mucous membranes*, the *cornea* and recently *burned or traumatized skin areas* are anesthetized by the topical application of the cocaine substitutes.

On the unbroken skin and mucous membranes the less soluble derivatives are frequently employed for a long continued action. Thus *ethyl aminobenzoate* and *butyl aminobenzoate* are employed respectively in 4 to 10 per cent ointment or powder or 2 per cent ointment for surface anesthesia or as an antipruritic.

The butyl preparation and procaine may cause an annoying *contact dermatitis* in sensitive individuals.

For *open wounds and ulcers* a pack or wet dressing of 1 to 2 per cent diothane may provide sufficiently effective local analgesia to permit the removal of foreign bodies, cleansing of the wound and approximation with a small suture.

In *ophthalmic practice* 2 per cent holocaine or 2 per cent butyn are excellent for surface anesthesia as in the removal of a foreign body or to allay the pain or blepharospasm that accompanies inflammations of the deeper structures of the eye or the administration of a painful astringent. In the *nose and throat* $\frac{1}{2}$ to 1 per cent pontocaine or 2 to 4 per cent procaine are employed for topical anesthesia.

In *genitourinary work* cocaine must never be used because of its rapid absorption through the mucous membranes. Local anesthesia prior to instrumentation (such as the passage of a catheter, a sound or a cystoscope) is effected by 2 to 4 per cent procaine, 2 per cent tetracaine or 2 per cent metycaine.

Infiltration Anesthesia

Infiltration anesthesia is the most commonly employed procedure for the production of local anesthesia.

Infiltration anesthesia is produced by the injection of 1 or 2 per cent procaine or some other of the soluble cocaine substitutes. A $\frac{1}{4}$ inch 27 gauge hypodermic needle attached to a hypodermic syringe of 2, 5, 10 or 20 cc capacity (preferably provided with a needle lock) is employed.

The local anesthetic solution may be purchased in sterile ampoules ready for use. For economy, tablets are provided. These may be dissolved in sterile distilled water to make $\frac{1}{4}$, 1 or 2 per cent concentration. The addition of one drop of 1:1000 epinephrine chloride to each 10 cc of cocaine substitute delays absorption, diminishes the possibility of toxic reaction, increases the duration of anesthesia and renders the field relatively bloodless by producing local vasoconstriction. Prepared ampoules and tablets containing the proper proportion of epinephrine are also available.

Thirty cc of a 2 per cent procaine and 125 cc of a 1 per cent solution may be introduced safely.

Technic—For infiltration anesthesia, intradermal wheals about 1 to $1\frac{1}{2}$ cm in diameter are raised by injection of the solution through a narrow gauge needle that is firmly attached to a syringe provided with a needle lock.

The area of anesthesia may be extended by raising multiple wheals throughout the length and breadth of the area to be anesthetized, or the needle may be directed intracutaneously along the line of the proposed incision. Longer needles are substituted for the $\frac{1}{4}$ inch needle if the area is particularly extensive.

When the surface or intracutaneous anesthesia has been accomplished, the smaller needles are discarded and 21 to 23 gauge 2, 3 or 4 inch needles are attached to the lock and the deeper structures are infiltrated in all directions. Approximately 1 cc is injected for each centimeter of tissue.

During the infiltration, the motion of the needle and the pressure on the plunger should be made continuously and simultaneously. Great care is exercised lest a small blood vessel be entered. If blood appears in the barrel upon withdrawal of the piston, the injection is terminated instantly.

Infiltrations are made with the greatest care in the region of the larger blood vessels and of bone. In the former instance, frequent aspirations should be performed to ascertain that the injection is not being made intravascularly. In the region of bone impingement upon the dense structure may dent the needle or actually cause it to break off.

Field Block

In field block, a wall of anesthesia is produced across the path of nerves supplying the proposed operative site. The subfascial layers are infiltrated first so that subcutaneous infiltrations do not produce distortion of the landmarks. See Fig. 1128 B.

and the spray directed at the operative site. The container is held at a distance of approximately 12 to 18 inches from the skin area. Shortly frosting occurs at the sprayed site and it is possible to make a small and superficial incision painlessly through the frozen area. This type of local anesthesia is satisfactory only for simple incisions since further operative work is not possible without the production of pain.

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tions are outside an infected zone (3) the wide area of anesthesia permits the use of retractors (4) better relaxation is gained particularly in the region of a sphincter as for example in rectal work

Nerve Block or Conduction Analgesia

Nerve block or conduction analgesia is the method of choice for procedures of any magnitude or those which require careful dissection (particularly of a digit) Nerve block requires more careful technique than the previously described methods It is necessary to have a precise knowledge of the location of the major nerve trunks See Fig. 1128 A and C

The most frequently employed blocks are *digital median* and *ulnar nerve block* at the wrist *caudal* or *epidural block* *subarachnoid block* or *spinal anesthesia* The expert may also produce blocks of the brachial plexus the cervical plexus certain of the cranial nerves and the dental



Fig 1129—Blocking the finger with a local anesthetic solution Anesthetic solution deposited close to the bone Location of anterior and posterior nerves should be kept in mind and a greater amount of solution deposited about location of the nerve

branches The latter procedures should be limited to the specialist anesthesiologist or one who is particularly familiar with the anatomy and the technique

Digital Block—An entire finger or toe can be anesthetized with as little as 3 cc of 2 per cent procaine distributed subcutaneously in a ring around the base of the digit Wheals are produced at the base of the digit on the medial and lateral aspects of the dorsum From these wheals an entire circumferential injection can be made using small hypodermic needles The injected area is massaged to avoid the production of dense edema which might constrict the digital vessels and impair the circulation of the tip of the extremity Following such an injection in about five minutes the entire digit becomes sufficiently insensitive to permit surgical procedure such as the incision of an infected area

Generally the field block is produced in the shape of a diamond the operative site corresponding to the pitcher's mound on a baseball field

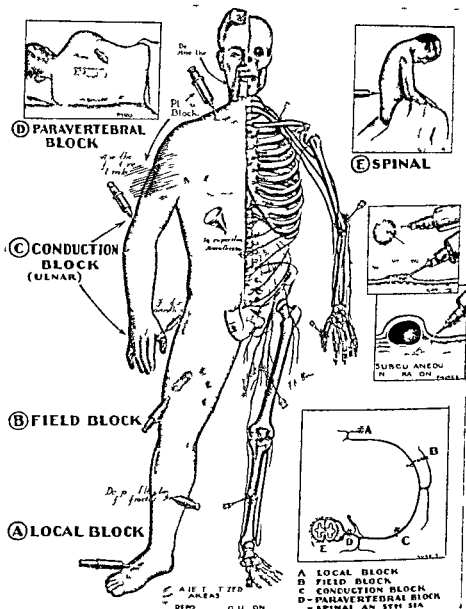


FIG. 1128.—Nerve block a study of basic methods showing on the two sides of the body the surface appearance and anesthetized areas and the course of the nerves and actual site of deposit of the anesthetic solution *

Field block of an extremity may be effective by infiltrating the lines above the surgical field since the nerve supply is entirely from one direction

The advantages of field block over infiltration are (1) The production of anesthesia without interference with the surface anatomy (2) inject

* Livingston in Christopher Textbook of Surgery

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The most frequently employed blocks are digital, median and ulnar nerve block at the wrist, caudal or epidural block, subarachnoid block or spinal anesthesia. The expert may also produce blocks of the brachial plexus, the cervical plexus, certain of the cranial nerves and the dental



Fig. 1129.—Blocking the finger with a local anesthetic solution. Anesthetic solution is injected close to the bone. Location of anterior and posterior nerves should be kept in mind and a greater amount of solution deposited about location of the nerves.

branches. The latter procedures should be limited to the specialist anesthesiologist or one who is particularly familiar with the anatomy and the technique.

Digital Block.—An entire finger or toe can be anesthetized with as little as 3 cc. of 2 per cent procaine distributed subcutaneously in a ring around the base of the digit. Wheals are produced at the base of the digit on the medial and lateral aspects of the dorsum. From these wheals an entire circumferential injection can be made using small hypodermic needles. The injected area is massaged to avoid the production of dense edema which might constrict the digital vessels and impair the circulation of the tip of the extremity. Following such an injection in about five minutes the entire digit becomes sufficiently insensitive to permit surgical procedure such as the incision of an infected area.

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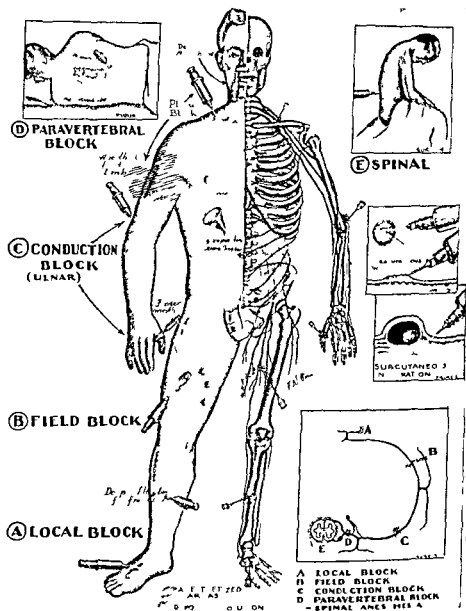


FIG. 1123.—Nerve block: a study of basic methods. Showing on the two sides of the body the surface appearance and anesthetized areas and the course of the nerves and actual site of deposit of the anesthetic solution.

Field block of an extremity may be effective by infiltrating the lines above the surgical field since the nerve supply is entirely from one direction.

The advantages of field block over infiltration are (1) The production of anesthesia without interference with the surface anatomy (2) injection

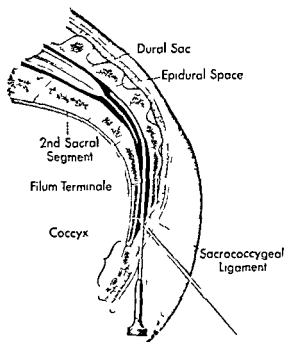


Fig 1130—Sagittal section of sacral region showing method of insertion of needle for caudal analgesia *

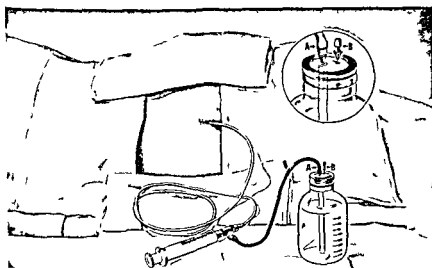


Fig 1131—Assembly for administration of continuous caudal analgesia. The needle attached to the short dark tubing through which the solution is to be withdrawn is inserted through the depression in the stopper centered below the glass tube (labeled A). Another needle is inserted through the other depression (labeled B) to allow ingress of air. Hose-hub needles such as those shown are not generally included with standard caudal analgesia apparatus.

Courtesy of Eli Lilly and Company

In the presence of cellulitis or of lymphangitis digital block must be avoided lest the infection spread. When the finger block is used a rubber band tourniquet should never be placed about the base of the finger. Epinephrine is not used with the anesthetic solution since further embarrassment of the local circulation may give rise to serious vascular damage culminating in gangrene.

Median and Ulnar Nerve Block at the Wrist—The vast majority of surgical procedures of the hand and fingers are concerned with the palmar aspect which is supplied by the median and ulnar nerves.

ANATOMIC REVIEW

The *median nerve* supplies the lateral two thirds of the palm of the hand the lateral aspect of the thumb the palmar aspect of the second and third fingers and the lateral half of the palmar aspect of the fourth finger as well as the dorsum of these fingers over the distal phalanx. The *ulnar nerve* supplies the medial half of the fourth finger the entire fifth finger as well as the palmar and dorsal aspects of the medial third of the hand. The *radial nerve* supplies the dorsal aspect of the lateral two thirds of the hand and the medial and dorsal aspects of the thumb. These areas alone may retain all sensation when median and ulnar nerve block is produced at the wrist.

The median and ulnar nerves are blocked at the wrist at the level of the ulnar styloid process. The patient is requested to flex the wrist while the anesthetist opposes the motion by holding the fingers. In this way the tendons of the flexor carpi radialis and of the palmaris longus are made prominent. At the level of the ulnar styloid process the median nerve lies just beneath the deep fascia in the interval between the two tendons. At the same site the ulnar nerve is blocked just to the radial side of the tendon of the flexor carpi ulnaris.

Median Block—Median block is executed in the following manner. The patient's arm rests flat on the operating table with the palm facing upward. An intracutaneous wheal is raised between the tendons of the flexor carpi radialis and the palmaris longus on a line passing through the ulnar styloid process.

The needle is introduced perpendicularly to the skin and advanced until it pierces the deep fascia. When the fascia is pierced the needle is advanced another half centimeter and the patient is requested to report the production of paresthesia. At this time 3 to 5 cc of 2 per cent procaine are injected without moving the needle.

It may be necessary where the nerves pursue an atypical course to withdraw the needle and insert it laterally beneath the tendon of the flexor carpi radialis until paresthesia is produced. If it is not possible to directly locate the nerve and produce paresthesia 5 to 6 cc of procaine may be distributed in the interspace between the tendons and beneath the tendon of the flexor carpi radialis. Care must be exercised not to pass the needle too deeply so that the injection is made into the capsule of the wrist joint for the anesthetic solution is valueless at this site.

After injection the area is massaged gently to accelerate diffusion of the anesthetic. The latter should produce its effect in 5 to 10 minutes.

Ulnar Block—Ulnar block is produced by raising a wheal on the radial side of the tendon of the flexor carpi ulnaris at the level of the ulnar styloid process. The needle is then inserted through the wheal and passed through the deep fascia. It is then directed somewhat medially so that its tip comes to lie beneath the tendon. If the nerve trunk is in the vicinity paresthesia will be produced and 3 cc of 2 per cent novocaine may be injected.

spinal fluid is permitted to drop from the needle directly into the ampoule containing the crystals. The crystals are dissolved in the spinal fluid by aspirating the material back and forth into a syringe. When solution has been completed the dose of procaine is decided. The excess of fluid is discarded and the remainder is reinjected intrathecally. Approximately 50 to 60 mg ($\frac{3}{4}$ to 1 grain) of procaine will provide sufficient anesthesia for any operation on the lower extremities or in the pelvis. Anesthesia will last from 45 minutes to one hour. For abdominal operations doses up to 200 mg (3 grains) of procaine may be employed.

After the injection of the local anesthetic solution has been accomplished the needle is withdrawn. The patient is turned flat on the back for from ten to fifteen minutes. After this he may be put in the Trendelenburg position.

Untoward Reactions—The larger doses of procaine will almost invariably be accompanied by a marked and at times serious fall in blood pressure. This drop may be anticipated and partially prevented by a preliminary subcutaneous injection of from 12 to 24 mg ($\frac{3}{16}$ to $\frac{3}{8}$ grain) of ephedrine.

The more serious dangers of spinal anesthesia include *meningitis* from bacterial contamination, *paraplegia* from a toxic myelitis and the occasional and unforeseen sudden fatality from *vasomotor or respiratory collapse*.

GENERAL ANESTHESIA

In an emergency it may be necessary to administer a general anesthetic in the office or in the home. This may be accomplished by intravenous anesthesia with the barbiturates or the use of ether, chloroform, vinyl ether or ethyl chloride.

Intravenous Anesthesia—The induction of general anesthesia by the intravenous use of the barbiturates previously was considered too risky for purposes of general practice. The Council on Pharmacy of the American Medical Association has recently approved of the use of pentothal sodium, one of the more soluble and rapidly acting barbiturates.

Pentothal sodium is supplied in ampoules containing 0.5 gm ($7\frac{1}{2}$ grains) and 1.0 gm (15 grains). Each ampoule also contains 30 or 60 mg ($\frac{1}{2}$ or 1 gr) of anhydrous sodium carbonate which acts as a buffer. Combination packages consist of one ampoule of the drug and one ampoule of sterile chemically pure water for making the solution.

The operator has the choice of making a $2\frac{1}{2}$ per cent or a 5 per cent solution. For the former the smaller ampoule is dissolved in 20 cc of the sterile chemically pure water for the 5 per cent solution 1 gm pentothal sodium is dissolved in 20 cc of sterile chemically pure water. The solution must be made up fresh and it cannot be allowed safely to stand for more than four hours.

Pentothal Sodium (N.N.R.) given by slow intravenous injection rapidly produces full surgical anesthesia that is of short duration. Heeding proper precautions the danger is small and sequels are absent so that the patient rapidly recovers and may leave the office.

Technic and Uses—Pentothal sodium must not be given by the operator. The anesthetist must be in constant attendance carefully observing the patient while the operator completes his technical procedures.

When it is necessary to produce a complete anesthesia of the hand including the distribution of the radial nerve a block anesthesia can be effected by making a ring of 1 per cent novocaine about the entire wrist in addition to performing median and ulnar block. Approximately 1 cc is injected in each centimeter of the subcutaneous tissue.

Caudal or Epidural Block—Caudal or epidural block effects analgesia of the *perineum urethra anus lower rectum prostate* and the distribution of the *sciatic nerve* and may be employed for surgical anesthesia or for the relief of sciatic pain.

In the production of caudal block the needle is passed through the *sacral hiatus* and the *anesthetic fluid* is deposited in the extradural space within the sacral canal.

To perform an epidural block the patient is placed on the right side. The sacral hiatus is identified by palpation as a depression at the junction of the coccyx and sacrum. An intradermal wheal is made over the hiatus and the subcutaneous tissues are infiltrated. The area is then massaged to remove the edema resulting from the injection so that the landmarks are again palpable.

A spinal puncture needle with stylet in place and bevel turned upward is introduced through the wheal in such a manner that it makes an angle of about 20 degrees with the skin. The point of the needle is directed cephalad. The sacrococcygeal membrane is pierced and the point of the needle impinges upon the anterior wall of the sacral canal. The needle is then slightly withdrawn and the hub is depressed towards the gluteal cleft so that the needle can be advanced gradually in the line of the sacral canal. Employing extreme precaution the needle is advanced in the canal for a distance of about 6 cm. The stylet is then withdrawn and the hub is observed to make certain that neither blood nor spinal fluid escapes since injection into a vessel or the spinal canal may lead to serious symptoms of cocaine poisoning.

If there is no flow from the needle a syringe filled with 2 per cent procaine is connected and gentle aspiration is performed to further ascertain that the tip of the needle is neither in a vein nor in the subarachnoid space. If all is well the procaine solution is injected very slowly employing a total of 30 to 40 cc. The needle is gradually withdrawn so that the anesthetic fluid is distributed throughout the length of the sacral canal. Anesthesia sets in slowly requiring about 15 to 20 minutes.

Subarachnoid Block or Spinal Anesthesia—Spinal anesthesia is readily performed by any practitioner capable of executing a lumbar puncture (p. 3782). For so called low spinal anesthesia the injection is preferably made in the third or fourth lumbar interspace. High spinal anesthesia may be administered in any of the other lumbar interspaces. *Low spinal anesthesia* is employed frequently in hemorrhoidectomy, operative cystoscopy and in the treatment of compound and complicated fractures of the lower extremities. *High spinal anesthesia* may be sufficient for the performance of any major procedure from the level of the xiphoid down to the toes.

Technic—For the induction of spinal anesthesia special ampoules containing pure crystals of procaine are employed. A lumbar puncture (p. 3782) is performed under strictly aseptic precautions. An ampoule of the pure crystals of procaine is opened and approximately 3 to 5 cc of

The fourth and fifth fingers are free to support the chin of the patient if it shows any tendency to fall. Petrolatum or mineral oil should be applied to the eyes as these anesthetics are locally irritating.

The anesthetic is dropped or sprayed on a mask at a rate determined by the substance employed. At first the anesthetic is added very slowly so that there is minimal irritation to the mucous membrane. Later the rate can be increased particularly during the period of excitement that characterizes the induction. During this phase of anesthesia the patient must be restrained by an assistant and the rate of administration may be increased so that the period of excitement is completed as rapidly as possible. However if the respiration becomes irregular and interspersed with long periods of apnea the mask should be raised since the first respiration after cessation will be quite deep and the danger of overdosage from a heavy concentration of vapor must be avoided.

If the patient begins to cough the rate of dropping should be decreased temporarily since the cough results from irritation of the mucous membranes by the strong concentration of vapors. The airways should be kept open by turning the patient's head to one side, extending the head and neck and holding the chin up with the fourth and fifth fingers of the hand that holds the mask. If the patient vomits the head is turned to one side and lowered to prevent vomitus and mucus from entering the airway.

Surgical Anesthesia—As the excitement subsides and the respiration becomes deep, regular and even the patient enters the stage of surgical anesthesia. This should occur in 8 or 10 minutes with ether, 5 to 8 minutes with chloroform and much more rapidly with ethyl chloride and divinyl ether or vinethene.

During surgical anesthesia the anesthetist regulates the depth of anesthesia by varying the rate of dropage. With ether the maintenance rate approximates one drop per second. With chloroform no more than 3 or 4 drops are required every 10 seconds. The anesthetic should be dropped on different portions of the mask to assure a more even distribution. When the excitement has abated and surgical anesthesia has been accomplished the anesthetist may check on the rate and depth of respiration, the pupillary reaction, eyeball movements, the pulse and blood pressure.

The phase of surgical anesthesia must be kept as smooth as is possible. A resumption of the period of excitement calls for deeper anesthesia by increasing the rate of flow.

Toxic Phase—The toxic phase of surgical anesthesia calls for prompt action. Under these circumstances intercostal activity ceases, the pupils dilate with loss of light reflex, the pulse rate becomes rapid and irregular and later weak and thready, respiration becomes shallow and may cease.

Overdosage with chloroform produces pallor, whereas a purple cyanosis indicates overdosage with ether or respiratory obstruction with any of the general anesthetics.

Treatment of Anesthetic Poisoning—At the first evidences of toxicity the anesthetic is stopped, the surgical procedure is discontinued, the patency of the airway is assured by inserting a tracheal tube or passing a catheter into the nostril, artificial respiration is instituted and a mixture of 90 per cent oxygen and 10 per cent carbon dioxide or pure oxygen is

When all of the preparations essential to the surgical procedure have been completed the anesthetist introduces a 20 gauge needle into an available and readily accessible vein. The patient is instructed to count while the pentothal (2½ per cent) solution is slowly injected intravenously. After the injection of 4 to 6 cc of the solution the needle is left in place but no further anesthetic is injected for a period of about 30 seconds. About this time the patient ceases to count and shows evidences of relaxation. The jaw drops and must be supported by the anesthetist to prevent respiratory difficulty.

If at the end of 30 seconds relaxation does not occur the injection is continued at a rate of approximately 1 cc every 15 seconds with a pause of 30 seconds after the injection of the next 1 or at most 2 cc. It is rarely necessary to inject more than 12 cc to produce satisfactory anesthesia. The presence of anesthesia is judged best by the alteration in the depth of respirations. The latter become shallow and regular without appreciable change in rate. The respirations must be watched carefully by the anesthetist since respiratory paralysis is a toxic symptom that requires emergency treatment.

The maximal dose in a single administration should not exceed 20 cc. The duration of the anesthesia may be from a momentary relaxation to a period as long as 20 to 30 minutes.

Intravenous anesthesia is useful for painful dressings, instrumentation such as *cystoscopy*, the manipulation of joints, incision of abscesses, implantation of radium and similar procedures.

Recovery is pleasant. The patient usually regains consciousness about 15 minutes after the cessation of a prolonged anesthesia, and in a few moments after cessation of short anesthesia. He may be drowsy for several hours after either prolonged or short anesthesia and may complain of some ataxia.

Untoward Reactions—Nausea and vomiting rarely occur. Induction is usually smooth without a period of excitement except in those who demonstrate an idiosyncrasy. The patient may be premedicated with atropine. Neither morphine nor its substitutes should be employed since they tend to increase the respiratory depression.

If respiratory depression occurs the anesthesia is stopped immediately and the intravenous needle is used for the introduction of 3 to 5 cc of coramine (p 3871) as a respiratory stimulant. Artificial respiration may then be practiced. If there is available a mixture of 90 per cent oxygen and 10 per cent carbon dioxide this should be administered.

Inhalation Anesthesia—In the office or home chloroform, ether, ethyl chloride and vinyl ether may be administered for general anesthesia by means of an open mask consisting of a wire mesh covered with a few layers of gauze.

Inhalation anesthesia should be given by a nurse, anesthetist or colleague except in a grave emergency when a member of the family may be called upon to maintain the anesthesia after the induction by the physician-operator. In general the physician should have at hand the apparatus and drugs necessary for any untoward reactions.

Induction—For the induction of anesthesia the patient reclines. The mask is held by the operator between his thumb, index and middle fingers.

chloride. Induction is rapid as is recovery and the risk of toxicity is more nearly that of ether than that of ethyl chloride.

THE SCOPE OF MINOR SURGERY

Planned minor surgery in the clean field includes such procedures as the injections of varicose veins, excision of a sebaceous cyst and the removal of a cutaneous neoplasm. It is performed at the convenience of patient and physician. There is small need for elaborate preoperative preparation, wound closure is a relatively minor problem, only a simple protective dressing is needed, and postoperative care is limited to the removal of sutures.

In deliberate minor surgery in a *contaminated field*, slightly more complicated problems arise. Closure of the wound may be unwise, local or systemic prophylactic chemotherapy may be advisable, frequent dressings are required, and complications are occasionally encountered.

The *minor surgery of trauma* focuses attention first upon the patient and then his wound. Accidental injuries are associated with anxiety, apprehension, varying degrees of emotional distress and shock. Often they are accompanied by blood loss, or continued hemorrhage, and there is of course inevitable contamination. The superficial lesion may overshadow or mask a much more significant abnormality in the deeper structures, as best exemplified by scalp and chest wounds. The presenting problems are much more complicated and difficult. Attention is directed first at the control of the most urgent disturbances which include shock and wound hemorrhage. The preoperative program includes the use of sedatives, analgesics, prophylactic chemotherapeutic agents and serum therapy. Exploration of the wounded area is primarily concerned with the detection of injury to structures more important than the subcutaneous and tegumentary tissues. There may be severance of tendons or nerves; there may be an underlying fracture; blood vessel integrity may have been sacrificed. Scalp injuries are a threat to the intracranial contents; chest and abdominal injuries jeopardize respectively the intrathoracic and intraperitoneal organs.

THE PLANNED MINOR SURGICAL ROUTE

Some of the planned minor surgical procedures are presented in the section on Treatment by Injection and Aspiration (p. 3770); others appear in specialty chapters such as Gynecology (p. 2522), Urology (p. 2297), Ophthalmology (p. 1554) and Disturbances of the Skeletal System (p. 2809). The remaining commoner injections and operations are as follows:

Excision of a Sebaceous Cyst or Neoplasm
Lymph Node Biopsy
Circumcision
Dorsal Split of Prepuce
Injection of Hydrocele
Vasectomy
Varicocelectomy
Injections of Varicose Veins
Ligation and Retrograde Injection of the Internal Sphenous Vein

Removal of an Ingrown Toenail
Excision of Pilonidal Sinus and Cyst
Injection of Hemorrhoids
Incision of Thrombosed External Hemorrhoids
Hemorrhoidectomy
Cauterization or Excision of Anal Fissure
Aspiration of a Joint or Bursa

begun by forced respiration if necessary. The intravenous administration of coramine may stimulate the respiratory center when toxicity is manifested by respiratory difficulty. It is a blunder to ply the patient with powerful drugs unnecessarily. The injection of caffeine, strychnine, digitalis or epinephrine is as apt to be noxious as helpful.

The sudden cessation of the heart beat such as may occur particularly with chloroform and ethyl chloride calls for massage of the cardiac areas through the diaphragm by inserting the fingers under the left costal border. If the situation becomes desperate the pulse beat imperceptible and the heart sound inaudible it is justifiable to inject intracardiacaally $\frac{1}{2}$ to 1 cc of 1:1000 epinephrine. The needle is plunged to the right of the sternal border in the third or fifth interspace. Blood is aspirated to assure entrance into the auricle and then the epinephrine is injected.

Recovery—In an uneventful general inhalation anesthesia the stage of toxicity is never reached. From the surgical phase the patient passes through a modified period of excitement similar to that following induction. Recovery occurs in a variable time.

The patient who receives an inhalation anesthesia should be kept at home or at the office for several hours. The irritation of the gas and the profuse perspiration may invite respiratory infection particularly pneumonia (p. 2189).

The Toxicity and Dangers of the Inhalation Anesthetics—Compared to local anesthesia the induction of general anesthesia is a distinctly hazardous therapeutic measure. In competent hands however the risk of intravenous anesthesia with the barbiturates is significantly less than that of the inhalation anesthetics.

Of the inhalation anesthetics *chloroform* and *ethyl chloride* are the most toxic and the most treacherous. With either of these preparations sudden death may occur without warning. Efforts at resuscitation however zealously executed may be quite futile. Additionally chloroform may cause later toxic effects referable to the heart muscle and liver parenchyma. As a result of these objections the use of chloroform has been all but abandoned except in obstetrical practice or where no other form of anesthesia is readily available.

Ethyl chloride is an unreliable and unpredictable anesthetic except in the hands of the most experienced. Many specialist anesthesiologists employ ethyl chloride for the induction of general anesthesia. These men recount uncomplicated experiences over the course of many years. The general practitioner who employs an occasional inhalation anesthesia should not however be guided by the experiences of the specialist. For his purposes ethyl chloride despite the advantages of rapid and easy induction with a brief and transitory phase of excitement is too dangerous for use since sudden respiratory or circulatory collapse may occur.

Ether is the safest of the inhalation anesthetics. The disadvantages of drop or open ether are the prolonged period of excitement during induction and the prolonged phase of recovery. In addition there is frequent nausea and vomiting following anesthesia and a tendency to the development of postoperative respiratory infections particularly pneumonia.

Amyl ether occupies a position midway between ether and ethyl

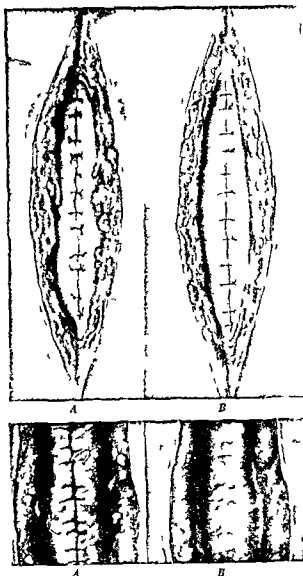


Fig. 113.—Experiments on dogs *A* and *B*. The appearance of the wounds at the completion of two rectus anastomoses made on the same dog. In *A* the tissues were roughly handled, mass ligatures were employed and the sutures in the anterior sheath of the rectus were tied very tightly. In *B* the tissues were handled with extreme gentleness, there were no ligatures of the fat and the sutures were tied very loosely. *A* and *B* represent the same wounds as they appeared five days later even though no clinical infection was present. In *A* note the necrosis produced by the ligatures and sutures—a veritable invitation to infection as well as a serious handicap to the healing of the wound.

skin from these heavy sutures. With deep tissue defects the combination of mattress sutures (Fig. 1134) of heavy material and superficial skin approximation with silk is usually satisfactory.

PREPARATION OF THE OPERATIVE SITE

The planned operative field is shaved when necessary using a safety or electric razor. In awkward areas such as the perineum a straight razor may be required. Mechanical cleansing of the field is accomplished by a vigorous scrub with soap and water using a brush or a cotton pledget. In cavities such as the vagina and rectum the soap is introduced on a ball of cotton held in a dressing forceps. After the soaping the field is cleansed by irrigation with water or saline solution. The importance of mechanical cleansing cannot be overemphasized; it is admittedly a time consuming nuisance but chemical sterilization is inadequate if the agent is placed on a dirty surface.

Following the saline flush the field is wiped with benzine, alcohol and ether and a wide area is painted with 3½ per cent Tincture of Iodine or 1:1000 solution Merthiolate N N R. The operative area is then draped and anesthesia is induced.

THE INCISION

While waiting for the local anesthetic to assume full potency the thoughtful practitioner plans his incision and considers his line of closure. A careless incision may result in a scar that is more objectionable than the original blemish. The incision is planned to lie along the lines of skin flexion unless it crosses an important deeper structure such as a main artery or nerve. The curved portion of the scalpel blade held perpendicular to the skin is employed for cutting purposes; beveled or chewed skin edges are difficult of approximation.

WOUND CLOSURE

The complete practitioner thinks in terms of approximation of structures before he wields his scalpel. Wound closure is relatively simple if there is no loss of tissue and can be accomplished by primary or secondary closure. With loss of tissue it may not be possible to approximate the edges unless a flap is employed. Wound healing and the appearance of the subsequent scar are largely determined by the nature of wound closure. Tissues that are roughly handled and brought together by tightly tied mass ligatures are apt to undergo necrosis with invitation to infection and ugly disfigurement. The gentle caressing of tissue, carefully planned ligatures that do not include fat and ties that are sufficiently loose to allow for the inevitable edema are rewarded by a hairline scar.

Primary Closure—Primary closure involves approximation of skin edges at the completion of the operative procedure. On the face and neck where a minimum scar is desired and the blood supply is plentiful the wound may be closed with flamed adhesive. The best type of suture is horsehair or Dermalon interrupted stitches laid ⅛ to ⅜ inch from the skin edge and tied with only slight tension to allow for edema. The stitches are placed a little less than ¼ inch apart. If the edges tend to retract deeper stitches of the traction type (Fig. 1133) are alternated but these should not be tied too tight lest small areas of skin become necrotic. Primary wound closure may also be accomplished with metal clips.

In areas such as the back where tissue approximation is more important than the appearance of the scar no 1 silk or steel wire is employed. Short lengths of rubber from discarded catheters may be used to protect the

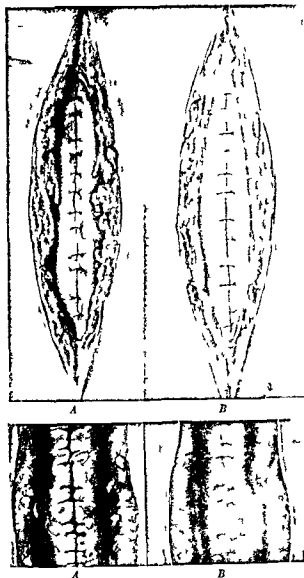


Fig 113 —Experiments on dogs *A* and *B*. The appearance of the wounds at the completion of two rectus neurectomies made on the same dog. In *A* the tissues were roughly handled, mass ligatures were employed and the sutures in the anterior sheath of the rectus were tied very tight. In *B* the tissue was handled with extreme gentleness, there were no ligatures of the fat and the sutures were tied very loosely. *A* and *B* represent the same wounds as they appeared five days later, even though no clinical infection was present. In *A* note the necrosis produced by the ligatures and sutures—a veritable invitation to infection as well as a serious handicap to the healing of the wound.

skin from these heavy sutures. With deep tissue defects the combination of mattress sutures (Fig 1134) of heavy material and superficial skin approximation with silk is usually satisfactory.

The subcuticular stitch is particularly adapted to areas of moderately loose skin in a scrupulously clean field. Number 0 plain catgut is usually used. The subcutaneous tissues are closed without tension using a continuous running stitch and bringing out the needle at one angle of the wound. The remainder of the suture is then laid into the corium of the skin edges by successive sweeps of the needle taking up the edge in bites about $\frac{1}{4}$ inch at a time. The accuracy of approximation may be checked after each suc-

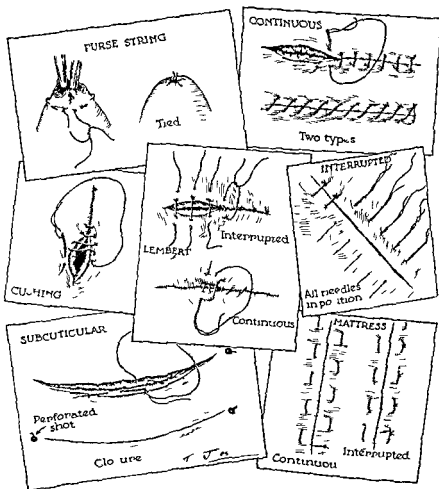


Fig. 1133.—Suture techniques

cessive bite by putting tension on the suture. The final stitch is taken at the far angle of the wound; the suture is then passed about 1 inch beyond the angle subcutaneously and brought out through the skin. The proper amount of tension is applied and held by a knot tied in the suture itself. After four days the knot and segment above the skin fall off and the wound is healed.

Secondary Closure—Secondary closure is a convenient method of prolonging the period of observation for twelve to twenty-four hours without

sacrifice of the advantage of suture approximation. At the time of injury or incision it may seem wiser not to close primarily because of possible bleeding or infection. Interrupted sutures of No. 1 silk or fine steel wire are introduced but not tied; picking is placed in the wound until the sutures can be tied with safety. For good approximation the skin edges are approximated within thirty hours.

Relaxing Incisions and Double Pedicles—Small defects from which a flap of skin has been completely avulsed can be closed if the skin and

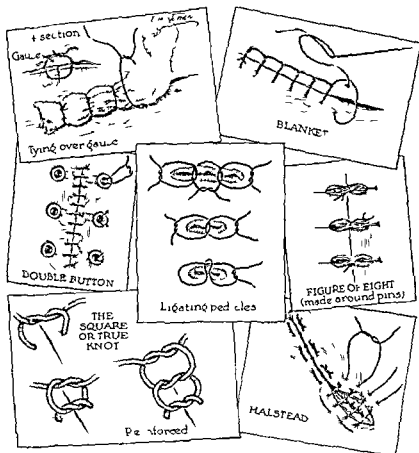


Fig. 1134.—Suture techniques.

subcutaneous tissues on either side of the defect are undermined for a moderate distance. The edges can then be mobilized and brought together over the defect without undue tension. If necessary, relaxing incisions can be made through skin and subcutaneous tissue parallel with the original incision. Thus the undermined tissues can be sutured together without tension. The resultant defect at the site of the relaxing incision can be closed by undermining the tissues further beyond it. The use of such a double pedicle flap permits tendons and nerves to be covered with a thick layer of skin and subcutaneous tissue and is preferable to a free graft.

Grafts—Larger defects that cannot be covered in this simple way can be managed by *free full thickness grafts* by *split thickness grafts* or by small *deep grafts* or *pinch grafts*

Free Full thickness Graft—The donor site is prepared by a soap and water scrub. Iodine is avoided since it causes damage to the underlying skin and affects its viability. The area to be grafted is measured carefully and an exact pattern cut from sterile paper or sterile cellophane. The pattern then is applied to the donor site and outlined on the skin with

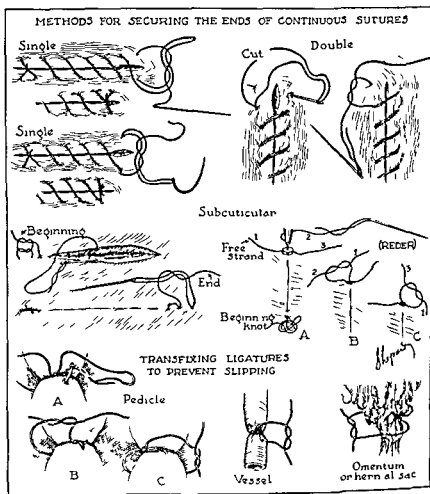


Fig 1135—Suture techniques

gentian violet solution. The graft is removed, the flap being raised with meticulous care. Bits of fat are carefully removed from the deep surface of the graft. The graft is handled gently. It should not be pinched with forceps. A good method for handling the flap is to place through the corners sutures whose ends are kept long. These act both as retractors and as handles to hold the flap. The site to be grafted is cleansed. Bleeding points are ligated. The flap is laid in place and sutured to the surrounding skin with many fine horsehair sutures placed along its margin.

Courtesy of Ethicon Suture Laboratories Johnson & Johnson

The graft itself is freely perforated at many points with a sharp scalpel in order to allow the escape of wound secretion. It is then covered with a layer of vaseline gauze or parestine mesh gauze followed by several fluffs, a rubber sponge and finally a snug bandage.

The entire region is immobilized in order to avoid any motion which might displace the graft from its bed. The wound should not be dressed for at least one week unless there is unmistakable evidence of infection such as pyrexia, persistent pain, lymphangitis or lymphadenitis. The

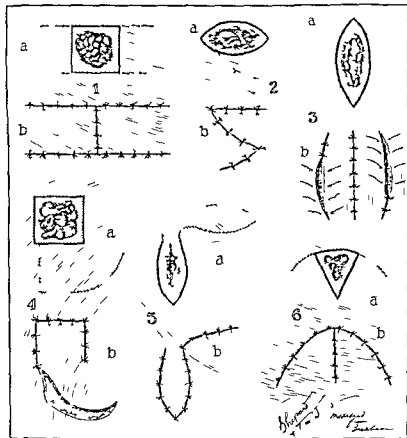


Fig. 1136—Sliding full thickness flaps

sutures are left in place for about 14 days. Prophylactic and antibiotic therapy using intramuscular injections of 50 000 units of penicillin every 3 or 4 hours greatly enhances the chances for a successful take.

Good free full thickness grafts for application to the palm or fingers and the finger tips can be obtained from the forearm. Clean amputations of finger tips are covered with such free grafts since otherwise healing is extremely slow and leaves a poor, painful scar.

Small deep grafts or pinch grafts give a less desirable cosmetic result than a full thickness graft. They are much harder, however, and will

survive even in the presence of slight infection. Preliminary preparation is the same as for any graft. The grafts themselves are obtained by elevating a small nubbin of skin using a fine needle. The apex of the pyramid thus created is cut off using a sharp razor or sharp scalpel. With the use of a needle the graft is laid in place. A sufficient number of grafts should be placed so that the area between them is of about the same diameter as the grafts themselves (approximately 0.5 cm in diameter). The grafts

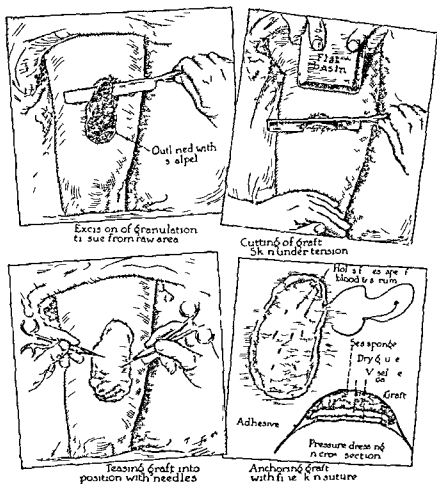


Fig 1137—Split skin grafts *

can be taken very close to each other leaving but tiny ridges of skin between the individual donor sites.

The entire grafted area is covered with paraffine mesh gauze, gauze fluff, and a snug bandage. It should be immobilized so that the individual grafts will not be wiped off with motion. The donor site can be covered by some vaselined gauze and a dry dressing. The dressing is not disturbed for about one week, at which time most of the graft will have taken and the donor site should be almost completely healed.

* Courtesy of Ethicon Suture Laboratories, Johnson & Johnson.

EXCISION OF SKIN SEGMENT

A segment of skin may be excised for a diagnostic biopsy in an obscure dermatosis or for the removal of a local lesion

- 1 Anesthetize by field block
- 2 Excise an elliptical area by clean dissection. Reserve specimen for pathologist
- 3 Obtain hemostasis by pressure if possible. Avoid deep ligatures
- 4 Close by primary suture. Use relaxation incisions or flap (p 2931) for wide areas
- 5 Apply vaselined gauze dressing
- 6 Observe after 48 hours
- 7 Remove sutures after 5 to 8 days
- 8 Maintain protective dressing an additional 2 or 3 days

EXCISION OF SPALFOUS CYST OR NEOPLASM

Removal of a superficial cyst or a benign neoplasm is readily performed under local anesthesia. Scalp lesions may require institutionalization for general anesthesia and expert assistance as cysts are often firmly and deeply adherent to the calvarium

- 1 Anesthetize by field block if in office. In hospital use intravenous or inhalation anesthesia
- 2 Make skin incision over or lateral to tumor to avoid penetration of capsule. Extend incision beyond limits of tumor at either end
- 3 Deepen incision to capsule
- 4 Separate capsule by blunt dissection. If necessary anchor tumor by grasping with blunt forceps or hemostat. Divide fibrous band with sharp scissors
- 5 Control bleeding by pressure if possible. Try to avoid deep ligatures
- 6 If capsule or cyst wall is ruptured attempt complete excision by dissection
- 7 Close by primary suture. Use interrupted stitches with silk or horse hair. Employ relaxation incision or flap if necessary
- 8 Apply vaseline gauze dressing
- 9 Observe after 48 hours
- 10 Remove suture after 5 to 8 days
- 11 Maintain protective dressing an additional 2 to 3 days

LYMPH NODE BIOPSY

The biopsy of a lymph node may be a more formidable problem than anticipated. Under ordinary conditions the procedure follows the technique of excision of a cyst or neoplasm. Unfortunately many difficulties are encountered. The node being diseased may be very adherent and vascular in the axilla and supraclavicular regions. Important structures are encountered requiring expert dissection under good light and with ample retraction. In the groin it is difficult to avoid contamination and infection.

Under these circumstances the practitioner may wisely admit the patient to the hospital for this procedure. Usually the magnitude of the suspected disease warrants observation and the pathologist evinces more

interest in the patient he has seen than one who is represented by a mere histologic slide

CIRCUMCISION

Circumcision is no longer a mere religious ritual but a procedure of election preferably performed by the obstetrician during the period of the puerperium. In the adult, circumcision is a painful operation and often requires a brief intravenous or inhalation anesthesia.

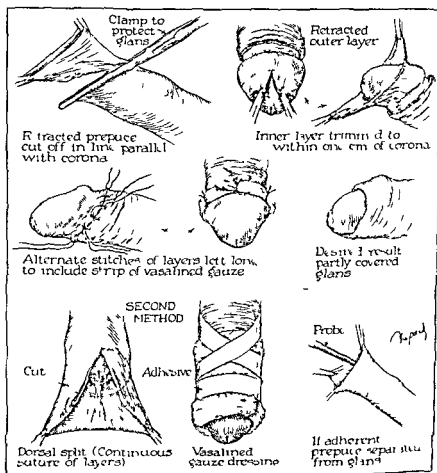


Fig. 1138—Circumcision

- 1 Inject a ring of local anesthesia around the base of penis
- 2 Free the adherent prepuce from the glans using a probe or director
- 3 Apply a clamp parallel to the corona and beyond the urethral orifice
- 4 Remove prepuce distal to clamp
- 5 Release clamp
- 6 Make circumferential skin incision beyond corona
- 7 Grasp fragment of mucosa and make dorsal slit between forceps
extend slit to within $\frac{1}{4}$ inch of corona

Courtesy of Ethicon Suture Laboratories Johnson & Johnson

- 8 Trim off mucosa leaving $\frac{1}{4}$ inch attached to corona
- 9 Suture coronal edge and penile edge. Do not tie sutures too tight. Leave alternate sutures long enough to tie over vaseline gauze dressing
- 10 Observe daily
- 11 Remove sutures after 3 to 5 days and apply dusting powder

DORSAL SLIT OF PREPUCE

A dorsal slit of the prepuce is required in the presence of edema usually due to a chancroid or lymphogranulomatous ulcer

- 1 Anesthetize as for circumcision
- 2 Grasp prepuce between forceps and make dorsal slit
- 3 Apply sulfonamide powder (p 99)
- 4 Apply vaseline gauze dressing

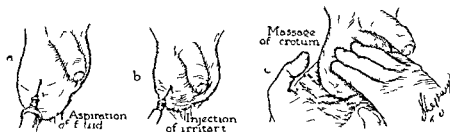


Fig 1139—Injection of hydrocele

INJECTION OF HYDROCELE

The hydrocele is clearly identified by transillumination since there is no interference with the penetration of the light rays. In his office the practitioner may attempt to obliterate the hydrocele cavity by aspiration of the fluid and the injection of sclerosing solution.

- 1 Carefully shave the surrounding area and induce local anesthesia of a section of the scrotum
- 2 Using a 24 gauge $1\frac{1}{2}$ inch needle attached to a lock syringe enter the hydrocele cavity and gently withdraw the hydrocele fluid
- 3 Leaving needle in place disconnect the aspirating syringe after the cavity has been emptied and substitute a 1 cc syringe containing a sclerosing fluid such as Solution of Inert Sugar N N R or Solution of Sodium Morrhuate (5 per cent) with benzyl alcohol (2 per cent) N N R
- 4 Inject the sclerosing solution leave needle in place for a few seconds and then gently massage to facilitate a thorough diffusion of the sclerosing solution
- 5 Put a collodion dressing over the site of the punctured wound of the scrotum

VASECTOMY

Vasectomy gives complete sterilization in the male. It prevents the development of a complicating orchitis following instrumentation of the urinary passages or prostatectomy.

- 1 Shave pubis and scrotum
- 2 Scrub thoroughly with soap and water and paint with Tincture of Merthiolate. Avoid iodine.

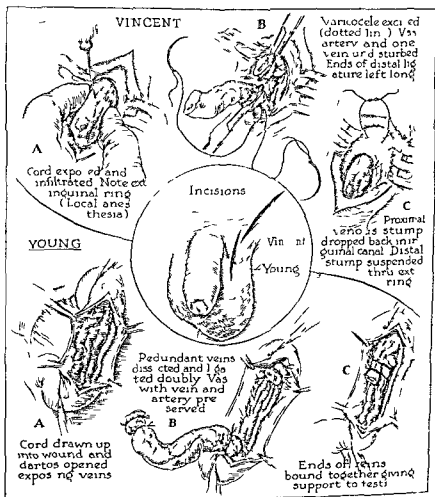


Fig 1140—Operations for varicocele (or varicocelectomy)

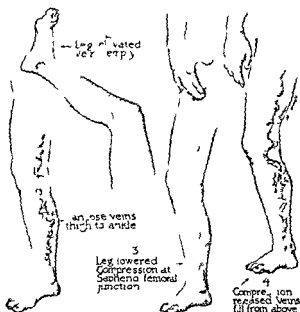
- 3 Identify the vas in the upper portion of the scrotum. It is a firm resilient whip like structure that can be rolled between thumb and index finger.
- 4 Hold the vas firmly in position between thumb and index finger anesthetize the overlying skin of the scrotum with 2 per cent procaine.
- 5 Incise the scrotum separate the tissues and isolate the vas.
- 6 Place a double ligature about the vas so that a section approximately 1 cm in length can be excised.

- 7 Replace the cut ends of the vas close the skin with No 0 cat gut and cover with a small collodion dressing
- 8 Repeat the operation on the opposite side
- 9 Inspect wound at end of 24 48 and 96 hours after which the sutures can be removed if necessary

VARICOCELECTOMY

Varicose veins of the scrotum may give rise to a dragging sensation or actual testicular pain They are readily excised under local anesthesia

- 1 Proceed as for vasectomy but when cord is exposed infiltrate with 2 per cent procaine



TRENDLENBURG TEST

Fig 1141—Trendelenburg test The leg is first elevated to empty the vein and constriction is applied Enough pressure is exerted to collapse only the superficial veins The patient then stands up while the pressure is maintained Note the collapse of the veins below the knee The fourth sketch shows the leg immediately after the pressure of the hand has been released Note the sudden filling of the veins from above *

- 2 Separate the vas the spermatic artery and one spermatic vein isolate the varicocele
- 3 Doubly ligate the veins of the varicocele and excise bind the cut ends together for a support for the testes
- 4 Close the wound and apply the collodion dressing as in vasectomy

INJECTIONS AND EXCISIONS OF VARICOSE VEINS

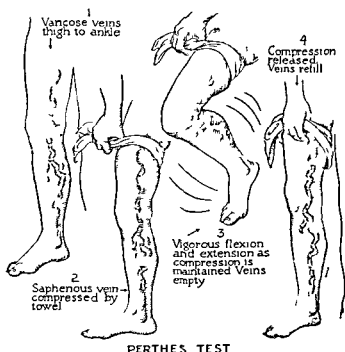
Varicose veins are most commonly seen in the distribution of the internal saphenous They are more frequent in women who have borne children

* de Takáts G in Christopher F Textbook of Surgery

dren and in those whose occupation requires long hours of standing with insufficient muscular exercise

Saphenous varicosities are ugly and many women demand treatment for cosmetic purposes. In other instances the disturbance produces local pain and cramps in the leg or a general asthenia. The varicose vein may rupture producing a severe hematoma. It may become infected giving rise to a protracted thrombophlebitis. It may cause trophic disturbances most particularly the development of the varicose ulcer.

Before instituting treatment of the varicose vein the hydrostatics of the problem requires investigation. The internal saphenous vein connects with the femoral vein at numerous points of the leg by means of communi-



PERTHES TEST

FIG. 1142.—The Perthes test. A tourniquet is applied with the patient in the standing position. Vigorous flexion and extension of the knee is carried out ten times. Note how the veins collapse after exercise indicating patency of the deep veins. When the pressure is released the veins suddenly fill from above.

cating vessels. It is necessary to determine the patency of the deep system and the competency of the vein valves particularly at the saphenofemoral junction before deciding the nature of the procedure.

The Trendelenburg Test—With the patient in the prone position the leg is extended to drain it of its pooled blood. A tourniquet is applied high up on the thigh with sufficient pressure to constrict the superficial but not the deep veins. The patient then stands while the constriction is maintained. After observation of blood flow in the vein for 20 to 30 seconds the tourniquet is removed and the reflux is observed.

The results of the Trendelenburg test are presented in chart form since the observations and their conclusions determine therapeutic indications.

de Takáts G. in Christopher F. Textbook of Surgery

Perthes Test—A tourniquet is applied to the region of the saphenous with sufficient tension to obliterate the superficial but not the deep vessels as in the Trendelenburg test. The patient is then requested to exercise the leg vigorously. The deep veins may be assumed to be patent if the superficial veins collapse; the patient does not experience pain in the calf and there is no demonstrable swelling in the foot and ankle. The deep veins may be assumed to be obliterated if the superficial veins do not collapse on exercise; the patient experiences pain in the calf of the leg and there is noticeable swelling of the foot and ankle. Under the latter circumstance attempts to obliterate the superficial saphenous system are ill advised.

Injection of Varicose Veins—An obliteration of the lumen of the varicose vein may be accomplished by injections of 20 to 30 per cent sodium chloride 15 per cent dextrose or 5 per cent sodium morrhuate containing 2 per cent of benzyl alcohol. The salt and sugar solutions may be injected in quantities up to 20 cc. but the morrhuate solution should not be admin-

TABLE 240.—THE TRENDLENBURG TEST

| <i>Tourniquet On</i> | <i>Tourniquet Off</i> | <i>Conclusion</i> | <i>Procedure Indicated</i> |
|--------------------------------------|---------------------------------------|---|----------------------------|
| <i>Veins fill slowly from below</i> | <i>Filling not accentuated</i> | <i>Veins dilated but valves competent</i> | <i>Injection</i> |
| <i>Veins fill rapidly from below</i> | <i>Filling not accentuated</i> | <i>Valves of communicating veins are incompetent</i> | <i>Injection</i> |
| <i>Veins fill slowly from below</i> | <i>Rapid filling from above</i> | <i>Saphenofemoral valve and valves of superficial veins are incompetent</i> | <i>Operation</i> |
| <i>Veins fill rapidly from below</i> | <i>Filling accentuated from above</i> | <i>Incompetence of all valves</i> | <i>Operation</i> |

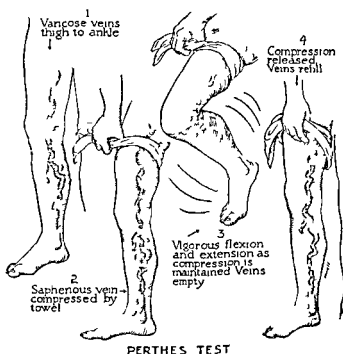
istered in an amount to exceed 5 cc. In any case short bevelled needles of 22 to 24 gauge are used with a 5 or 10 cc. syringe fitted with a lock.

- 1 With the patient standing cleanse the injection site gently by rubbing with alcohol.
- 2 Introduce the needle into the varicosity avoiding vessels above the knee and aspirate blood into the syringe.
- 3 With the second and third fingers of the free hand strip a segment of the vein 2 or 3 cm. long.
- 4 Maintain compression so that the segment of vein is emptied of blood. Inject the sclerosing solution leaving the needle in situ for 30 seconds. Withdraw the needle and maintain pressure with a small pad of gauze or cotton firmly taped into position for at least 48 hours.
- 5 Discontinue injection if patient complains of pain. Leave needle in place withdraw 5 cc. of blood and inject into painful area.
- 6 Remove the dressing and inject another segment of vein until the entire varicosity has been obliterated.

dren and in those whose occupation requires long hours of standing with insufficient muscular exercise

Saphenous varicosities are ugly and many women demand treatment for cosmetic purposes. In other instances the disturbance produces local pain and cramps in the leg or a general asthenia. The varicose vein may rupture producing a severe hematoma. It may become infected giving rise to a protracted thrombophlebitis. It may cause trophic disturbances most particularly the development of the varicose ulcer.

Before instituting treatment of the varicose vein the hydrostatics of the problem requires investigation. The internal saphenous vein connects with the femoral vein at numerous points of the leg by means of communi-



PERTHES TEST

Fig. 1142—The Perthes test. A tourniquet is applied with the patient in the standing position. Vigorous flexion and extension of the knee is carried out ten times. Note how the veins collapse after exercise indicating patency of the deep veins. When the pressure is released the veins suddenly fill from above.*

cating vessels. It is necessary to determine the patency of the deep system and the competency of the vein valves particularly at the saphenofemoral junction before deciding the nature of the procedure.

The Trendelenburg Test—With the patient in the prone position the leg is extended to drain it of its pooled blood; a tourniquet is applied high up on the thigh with sufficient pressure to constrict the superficial but not the deep veins. The patient then stands while the constriction is maintained. After observation of blood flow in the vein for 20 to 30 seconds the tourniquet is removed and the reflux is observed.

The results of the Trendelenburg test are presented in chart form since the observations and their conclusions determine therapeutic indications.

* de Takáts, G. in Christopher, F. Textbook of Surgery

Perthes Test—A tourniquet is applied to the region of the saphenous with sufficient tension to obliterate the superficial but not the deep vessels as in the Trendelenburg test. The patient is then requested to exercise the leg vigorously. The deep veins may be assumed to be patent if the superficial veins collapse, the patient does not experience pain in the calf and there is no demonstrable swelling in the foot and ankle. The deep veins may be assumed to be obliterated if the superficial veins do not collapse on exercise, the patient experiences pain in the calf of the leg and there is noticeable swelling of the foot and ankle. Under the latter circumstance attempts to obliterate the superficial saphenous system are ill advised.

Injection of Varicose Veins—An obliteration of the lumen of the varicose vein may be accomplished by injections of 20 to 30 per cent sodium chloride 10 per cent dextrose or 5 per cent sodium morrhuate containing 2 per cent of benzyl alcohol. The salt and sugar solutions may be injected in quantities up to 20 cc but the morrhuate solution should not be admin-

TABLE 210—THE TRENDLENBURG TEST

| Tourniquet On | Tourniquet Off | Conclusion | Procedure Indicated |
|-------------------------------|--------------------------------|--|---------------------|
| Veins fill slowly from below | Filling not accentuated | Veins dilated but valves competent | Injection |
| Veins fill rapidly from below | Filling not accentuated | Valves of communicating veins are incompetent | Injection |
| Veins fill slowly from below | Rapid filling from above | Saphenofemoral valve and valves of superficial veins are incompetent | Operation |
| Veins fill rapidly from below | Filling accentuated from above | Incompetence of all valves | Operation |

istered in an amount to exceed 5 cc. In any case short bevelled needles of 22 to 24 gauge are used with a 5 or 10 cc syringe fitted with a lock.

- 1 With the patient standing cleanse the injection site gently by rubbing with alcohol.
- 2 Introduce the needle into the varicosity avoiding vessels above the knee and aspirate blood into the syringe.
- 3 With the second and third fingers of the free hand strip a segment of the vein 2 or 3 cm long.
- 4 Maintain compression so that the segment of vein is emptied of blood. Inject the sclerosing solution leaving the needle in situ for 30 seconds. Withdraw the needle and maintain pressure with a small pad of gauze or cotton firmly taped into position for at least 48 hours.
- 5 Discontinue injection if patient complains of pain. Leave needle in place withdraw 5 cc of blood and inject into painful area.
- 6 Remove the dressing and inject another segment of vein until the entire varicosity has been obliterated.

Ligation and Retrograde Injection of the Saphenous Vein—When there are indications that the sapheno femoral valves are incompetent or when the varicosity is above the level of the knee it is far wiser to ligate the saphenous vein. If Perthes and Trendelenburg tests reveal incompetence of communicating veins in addition to sapheno-femoral incompetence the involved communicating veins must be ligated at the same time.



Fig. 1143—Varicose vein injection—standing position. No tourniquet is used.

- 1 Prepare the patient with 50 mg ($\frac{3}{4}$ gr) of demerol 0.4 mg ($\frac{1}{160}$ gr) of scopolamine hydrobromide injected 1½ hours before the anticipated procedure and 0.2 gm (3 gr) of sodium orally three quarters of an hour later.
 - 2 Infiltrate the skin in the region of the fossa ovalis with 2 per cent procaine.
 - 3 Expose the saphenous vein through a transverse incision and locate the saphenofemoral junction.
 - 4 Identify and isolate the three main branches of the internal saphenous vein.
- Riddle, Injection Treatment.

nous which include the superficial circumflex iliac the superficial external pudendal and the superficial inferior epigastric

- 5 Ligate and divide the branches
- 6 Ligate and divide the saphenous vein as close as possible to the femoral junction leaving a reasonable stump
- 7 Place a transfixion ligature through the saphenous vein stump

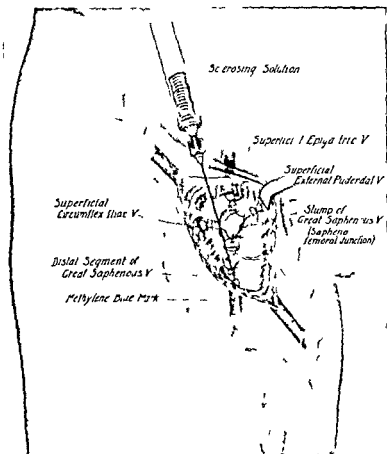


Fig 1144—Technic for ligation of great saphenous vein and its superior branches

- 8 Insert a ureteral catheter into the distal stump and introduce for a distance of 60 cm
- 9 As the ureteral catheter is slowly withdrawn instill sclerosing solution throughout the length of the vein
- 10 Ligate the distal stump and fix with a transfixion suture
- 11 Close the wound without drainage
- 12 Dress the wound carefully. Apply a compression bandage from toes to mid thigh
- 13 Have the patient walk to aid in the distribution of the sclerosing solution and encourage frequent weight bearing

REMOVAL OF INGROWING TOENAIL

The ingrown toenail is one of the more common nuisances of office practice. Removal is simply performed with gratifying results.

- 1 Induce a nerve block of the toe by the injection of 1 or 2 cc of 2 per cent procaine on each side
- 2 When anesthesia seems satisfactory apply a tourniquet to the base of the toe
- 3 Incise the mesial aspect of the toe parallel to the edge of the nail extend the incision from the tip of the toe to a point approximately 2 cc beyond the nail fold
- 4 Reflect a paronychia and an eponychial flap
- 5 Make a secondary incision through the nail the nail bed and the matrix

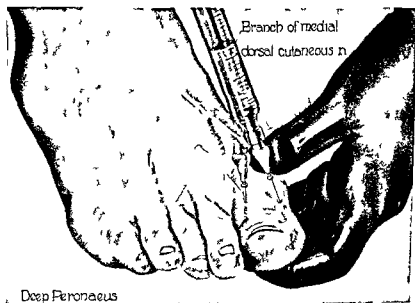


Fig 1145—For removal of toenail or other operative procedures on the great toe anesthesia is produced by depositing 4 to 6 cc of a 1 per cent novocain solution about its base

- 6 Excise the ingrowing portion of the nail and curette the proximal lateral angle of the wound to assure complete removal of the matrix
- 7 Apply a light packing of petrolatum gauze suture the flaps loosely with a single ligature
- 8 Observe the wound at 24 48 and 72 hours
- 9 Remove the packing and permit healing

EXCISION OF PILONIDAL SINUS

Excision of the pilonidal sinus and cyst may turn out to be a much more formidable procedure than anticipated. At times the lesion extends down to the sacrum or coccyx. If it becomes necessary for the practitioner to attempt removal of the pilonidal cyst without reference to the consult

ant surgeon he should insist upon hospitalization so that he has the advantage of a trained assistant. General anesthesia is required since infiltration distorts the local appearance and spinal injections are contraindicated by the near presence of infection.

- 1 Inject the pilonidal sinuses with methylene blue so that a guide is available for dissection

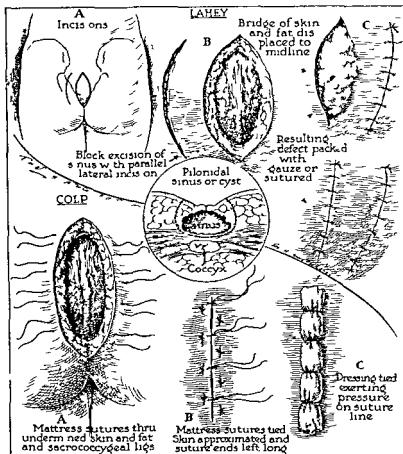


Fig. 1146.—Operative treatment of pilonidal sinus

- 2 Make oval incision around the area of the cyst and an accessory incision approximately two centimeters lateral to one side
- 3 Carefully excise the cyst, its wall and a thin layer of the surrounding areolar tissue
- 4 Introduce 2 or 3 gm. (30 or 45 gr.) of sulfanilamide powder (p. 99)
- 5 Draw skin together with interrupted steel wire sutures
- 6 Close accessory incision
- 7 If skin cannot be drawn together or there is gross infection, pack wound open and apply adhesive tape dressing

- 8 Dress duly Graft may be required if approximation cannot be maintained Give prophylactic intramuscular injections of 50 000 units of penicillin every 3 or 4 hours

INJECTION OF HEMORRHOIDS

Internal hemorrhoids unassociated with the external variety and showing no tendency to prolapse can be adequately treated by injection therapy

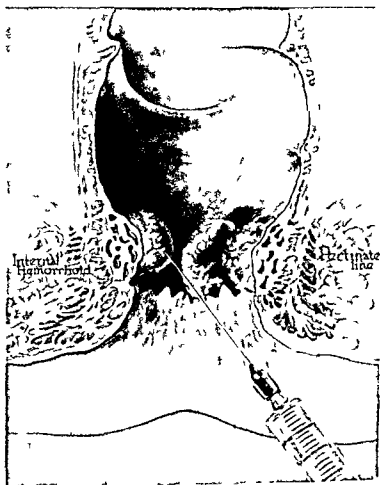


Fig 1147—Technic for the injection treatment of internal hemorrhoids

- 1 Following a cleansing enema the hemorrhoid is visualized by an anoscope with an angulated tip which permits one hemorrhoid to prolapse into the lumen
- 2 Fill a 2 cc syringe with 5 per cent quinine-urea hydrochloride solution
- 3 Using a special 2 or 3 inch needle sheathed throughout its entire length except for the distal one half inch prepare for injection therapy

Riddle Injection Treatment

- 4 Paint the site of injection with Tincture of Merthiolate
- 5 Insert the needle edge slightly proximal to the most prominent portion of the hemorrhoid. Keep the needle within the submucous tissue and do not enter the lumen of the vein. It should not be possible to aspirate blood into the syringe.
- 6 Inject 1 or 2 cc of the quinine solution. Use a sufficient quantity to distend the mucosa but not enough to produce blanching.
- 7 Remove the anoscope and prepare for further injections within a few days. Deposit 300 000 units of penicillin in oil twice daily.

INCISION OF THROMBOSED EXTERNAL HEMORRHOIDS

The thrombosed external hemorrhoid is frequently observed as a cherry red firm and extremely tender mass. Rapid relief of pain is obtained by making an incision and turning out the clot.

- 1 Infiltrate the base of the hemorrhoid with 1 per cent procaine.
- 2 Make a radial elliptical incision over the most prominent portion of the thrombosed hemorrhoid. Be careful to preserve the fibers of the sphincter.
- 3 Deepen the incision until the clot is exposed. Preparatory to evacuation.
- 4 Deposit 300 000 units of penicillin in oil twice daily. Caution the patient to avoid evacuation for 48 hours.

HEMORRHOIDECTOMY

The removal of hemorrhoids is required when conservative and injection treatments fail to give relief. The procedure should not be attempted unless the patient is hospitalized and assistance is accessible.

- 1 Using preoperative medication and low spinal anesthesia dilate the rectal sphincter so that at least four fingers can be inserted into the rectum.
- 2 Prepare the field by painting with Tincture of Merthiolate.
- 3 Grasp a hemorrhoidal mass with a triangular or hemorrhoidal clamp and draw it upward from the rectum.
- 4 Deposit 300 000 units of penicillin in oil twice daily. Caution the patient to avoid evacuation for 48 hours.
- 5 Make an incision midway between the clamp at the anal margin and the base of the hemorrhoid.
- 6 Deepen the incision until the hemorrhoidal vein is reached and then apply a large curved crushing clamp over the base of the hemorrhoid.
- 7 Place a transfixion suture at the apex of the hemorrhoid and tie.
- 8 Cut off the hemorrhoidal mass flush with the clamp using a scalpel.
- 9 Sew one end of the transfixion suture over the clamp.
- 10 Withdraw the clamp and tie the entire transfixion suture but avoid inclusion of the skin. Leave a small hiatus to provide drainage of the hemorrhoidal bed.
- 11 Repeat this procedure for each of the hemorrhoidal masses. Apply sulfanilamide powder and insert a thin strip ofaseline gauze into the wound. Apply a sterile gauze bandage held in place by a

- 8 Dress daily Graft may be required if approximation cannot be maintained Give prophylactic intramuscular injections of 50 000 units of penicillin every 3 or 4 hours

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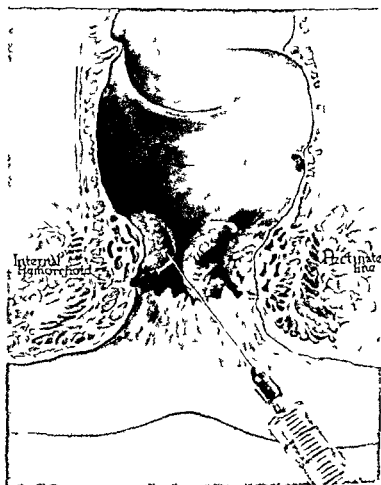


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- 1 Using preoperative medication and low spinal anesthesia dilate the rectal sphincter so that at least four fingers can be inserted into the rectum
- 2 Prepare the field by painting with Tincture of Merthiolate
- 3 Excise ulcer sentinel papilla sentinel pile and a wedge of perianal skin $\frac{1}{2}$ inch wide and 1 inch long
- 4 Incise radially superficial fibers of subcutaneous sphincter in midline posteriorly Follow post-operative hemorrhoidectomy routine (p 3947)

ASPIRATION OF A JOINT

Joint aspiration is practiced for diagnostic purpose but also to relieve pressure and remove extravasated blood in the attempt to guard against the later development of ankylosis

- 1 Using strict aseptic technic thoroughly scrub the area and paint with $3\frac{1}{2}$ per cent iodine solution

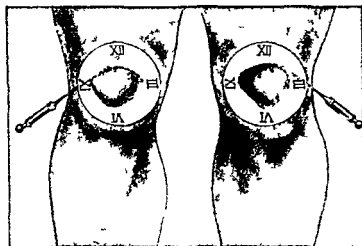


Fig 1149—Face-to-the-clock method of aspiration in synovitis of knee

- 2 Raise a wheal with 2 per cent procaine solution at the projected puncture point
- 3 For aspiration of the knee joint the needle is inserted 1 inch to the outside of the patella and is then carried toward the middle of the joint For entrance at the hip joint the injection is made midway between the great trochanter and the intersection of the femoral artery with Poupert's ligament The shoulder joint is approached from the posterior aspect the needle being inserted just distal to the base of the acromion process behind the posterior border of the deltoid and the tendon of the infraspinatus
- 4 Using an 18 gauge $1\frac{1}{2}$ inch needle attached to a lock syringe the cavity of the joint is entered gently and fluid is withdrawn
- 5 After withdrawal of the needle a pressure bandage is applied and maintained for at least 24 hours

T binder Deposit 300 000 units of penicillin in oil intramuscularly twice daily

- 12 After 12 hours remove the gauze and replace with a wet dressing containing warm witch hazel or boric acid solution
- 13 Avoid evacuation for at least 48 hours by using a low residue diet and small doses of Tincture of Opium

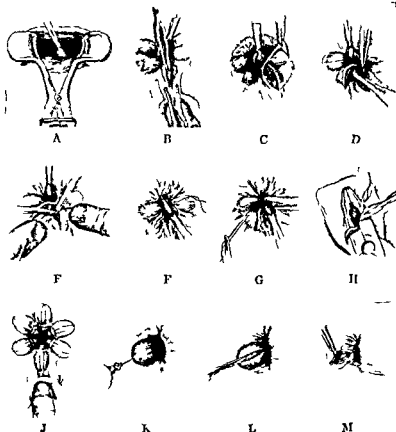


Fig 1148.—Hemorrhoidectomy *A* Dilator in place and internal hemorrhoid grasped with forceps *B* Skin over external hemorrhoid elevated with tissue forceps *C* *D* Veins are dissected from hemorrhoid *E* Clamping and ligating large bleeders *F* Transfixion suture at base of internal hemorrhoid tied *G* Amputation of hemorrhoid distal to ligation *H* Alternate method using hemorrhoid clamp and cautery *J* Dotted lines indicate scheme of skin excision following radiating lines *K* Injection of local anesthetic *L* *M* Method of closure of skin defect by interrupted sutures *

- 14 At the end of 48 hours give a retention enema of four ounces of warmed olive oil and assist the patient to attempt an evacuation
- 15 Following evacuation sitz baths are ordered two or three times daily

CAUTERIZATION OR DISSECTION OF ANAL FISSURE

The anal fissure is an excruciatingly painful affliction that requires operative relief

Courtesy of Ethicon Suture Laboratories Johnson & Johnson

missible Many ask for a drink of whiskey this too is quite in order provided that a note is made that the alcoholic beverage was taken after the accident occurred since the influence of drink may give rise to medico legal implications

RESTORATION OF BLOOD VOLUME

If there is a history of considerable blood loss and the patient appears exsanguinated preparations are made for a transfusion of 500 cc of *citrated blood* at the earliest possible moment The diminution in the red cells may be clouded by the hemoconcentration due to shock hence the history and clinical appearances are of greater significance than the actual erythrocyte count If the patient appears to be actively bleeding from some internal source hospitalization is mandatory since there may be some grave internal injury such as hemothorax or a rupture of spleen liver or kidney

PREVENTION OF INFECTION

In traumatic surgery each wound is either contaminated or infected The *contaminated wound* is defined arbitrarily as one in which soiling has occurred no longer than six hours before observation by the physician The *infected wound* is one in which there is evidence of active growth of bacteria tissue invasion and tissue destruction Infection is revealed by local and systemic manifestations The local phenomena include the cardinal signs of inflammation these may be confined to the cellular tissues the adjacent lymphatic vessels or nodes The systemic features are those of malaise and weakness demonstrable elevation of temperature and pulse rate and a leukocytosis

The systemic management of contamination and infection is accomplished by prophylactic chemotherapy serotherapy or both

Prophylactic Chemotherapy and Serotherapy—The progression from a contaminated to an infected wound with systemic manifestations places the practitioner in the position where he cannot defend himself if he has neglected to give prophylactic therapy It is the greater part of wisdom to administer precautionary treatment in a hundred unnecessary instances rather than to be culpable of withholding a life saving remedy in any one The experiences of war have clearly demonstrated the advantage of giving intramuscular injections of 50 000 units of *penicillin* (p 106) at four hour intervals Wounds that are grossly contaminated with soil or which have become fouled with human or animal excreta require the injection of *tetanus gas gangrene antitoxin* (p 297)

Curative Chemotherapy—In the face of obvious infection the practitioner does not hesitate to inaugurate systemic therapy He may employ a *sulfonamide* or *penicillin* in the presence of severe progressive infection the two agencies are effectually combined The ambulatory patient is given an initial dose of 2 to 4 gm (30 to 60 gr) of *sulfadiazine* with the usual precautions (p 88) or intramuscular deposits of 300 000 units of *penicillin* in oil The hospitalized patient may be treated with injections of *penicillin* in addition to the sulfonamide or as a substitute *Bed rest* is mandatory A normal diet is permitted *Fluids* are forced for purposes of diluting the urine in the presence of sulfonamide therapy and for detoxification

THE MINOR SURGICAL ASPECTS OF TRAUMA CONTAMINATION AND INFECTION

In the management of trauma contamination and infection the practitioner is confronted with many more complex problems than are met in the performance of deliberate or planned surgery. *First consideration* is given to the presenting or potential systemic effects of the local process. Unless there is active bleeding or intense pain the wound is *secondarily* considered after the broader implications have been cared for or anticipated.

The generic problems relative to the welfare of the patient include

- 1 The prevention or active treatment of shock
- 2 The production of analgesia and sedation
- 3 The restoration of blood volume
- 4 The prevention of infection
- 5 The detection and management of injury to the deeper tissues

PREVENTION OR ACTIVE TREATMENT OF SHOCK

The problem of shock is elsewhere reviewed in great detail (p 928). For present purposes it is sufficient to emphasize that shock accompanies each traumatic experience. It is a wise practitioner who anticipates difficulty even at the expense of unnecessary therapy for a majority of his traumatized patients.

At the first positive evidences of the actual presence of shock a *plasma infusion* is inaugurated. Injections of 1 cc of 1 per cent *neosynephrine* are given at 30 to 60 minute intervals and *analgesics* and *sedatives* are administered in liberal dosage. Provisions are made to supply *oxygen* by the *BLB mask using the oro nasal method so as to insure a concentration of 50 to 60 per cent*. Preparations are made for the removal of the patient to a hospital at the earliest safe moment. An effort is made to obtain hematocrit readings so that the blood concentration can be kept below 45 per cent by continuous or repeated infusions of plasma and blood.

ANALGESIA AND SEDATION

The traumatized patient must be made comfortable by medication with analgesics and sedatives. This principle is not dictated wholly in the interest of comfort but it is of signal importance in the prophylaxis of shock. Even if pain is only moderately severe a subcutaneous injection of *morphine sulfate* 15 mg ($\frac{1}{4}$ grain) *codeine phosphate* 60 mg (1 grain) *dilaudid* 2 mg ($\frac{1}{30}$ grain) or *demerol* 100 mg ($1\frac{1}{2}$ grains) is clearly in order with a repetition within an hour if needed. In the presence of severe anguish the dose of *morphine* can be given intravenously with almost instantaneous relief.

Whether or not the hypodermic injection is given the patient is entitled to an oral dose of a barbiturate such as *phenobarbital sodium* 30 mg ($\frac{1}{2}$ grain) or a hypnotic capsule such as *seconal sodium* 100 mg ($1\frac{1}{2}$ grains). This supplementary medication has the twofold purpose of lessening the central effects of the local anesthetic and of easing the strain of the situation. Most patients request a cigarette which is certainly per

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Emergency Treatment of Injuries to the Deeper Structures

The practitioner in the isolated community must be equipped to carry out emergency measures in the presence of injuries to the deeper structures. These procedures include tenorrhaphy, neurorrhaphy, repair of a laceration of the joint capsules, reduction of dislocations and fractures, amputations, relief of a tension pneumothorax, evacuation of blood clot from the neck, the care of a sucking wound of the chest, emergency cyst

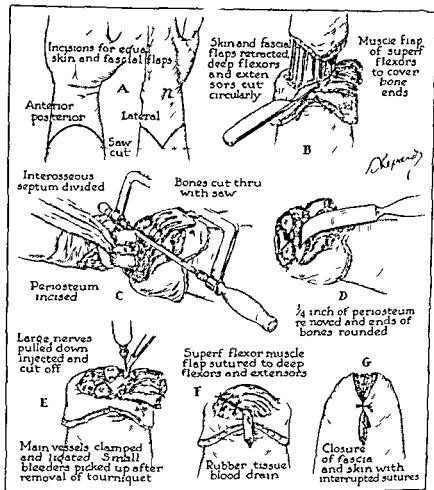


Fig. 1151—Amputation of forearm *

tostomy and tracheotomy In briefest outline these operations are sketched in the succeeding paragraphs but the physician is urged to consult standard practices of surgery for more complete information

Tenorrhaphy—With loss of motion indicating a tendon severance the practitioner arranges to give his patient full anesthesia. The wound is then explored with retraction and a good light to identify tendon ends and effect their union by sutures. The patient is kept under observation for a period of at least 48 hours during which time 50,000 units of penicillin are injected every 4 hours

* Courtesy of Ethicon Suture Laboratories Johnson & Johnson

Neurorrhaphy—The problem of nerve severance is managed in the manner of the divided tendon. The divided nerve ends may retract for a considerable distance in an extremity but an approximating suture as illustrated should prove successful in the restoration of function.

Repair of Laceration of the Joint Capsule—If there is obvious laceration of the joint capsule repair should be attempted by suture with 00 plain catgut. An exception to this rule exists in the human bite which is usu-

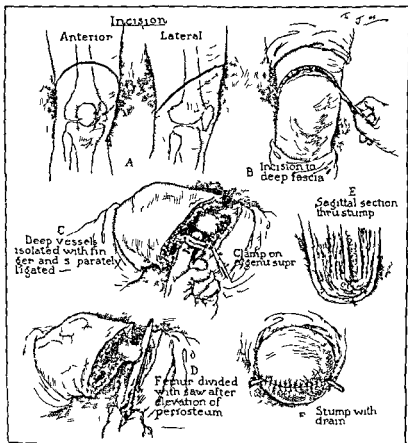


Fig 1152—Amputation of lower extremity above the knee

ally situated over the knuckle. So great is the danger of contamination and infection that this injury is best left open.

Reduction of Dislocations and Fractures.—See *The Skeletal System* (p 2964-2982)

Amputation—If it is necessary to perform an emergency amputation incisions are planned as indicated in the illustrations. Blood vessels are identified and firmly ligated. Nerve trunks are left long and injected with alcohol. Skin and fascial flaps are preserved but not closed at the scene of the accident. Sulfonamide powder is dusted liberally. Measures are insti-

Courtesy of Ethicon Suture Laboratories Johnson & Johnson

Emergency Treatment of Injuries to the Deeper Structures

The practitioner in the isolated community must be equipped to carry out emergency measures in the presence of injuries to the deeper structures. These procedures include tenorrhaphy, neurorrhaphy, repair of a laceration of the joint capsules, reduction of dislocations and fractures, amputations, relief of a tension pneumothorax, evacuation of blood clot from the neck, the care of a sucking wound of the chest, emergency cyst

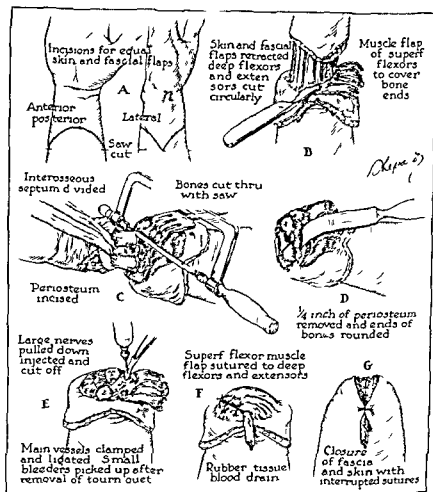


Fig 1151—Amputation of forearm

tostomy and tracheotomy. In briefest outline these operations are sketched in the succeeding paragraphs but the physician is urged to consult standard practices of surgery for more complete information.

Tenorrhaphy—With loss of motion indicating a tendon severance the practitioner arranges to give his patient full anesthesia. The wound is then explored with retraction and a good light to identify tendon ends and effect their union by sutures. The patient is kept under observation for a period of at least 48 hours during which time 50,000 units of penicillin are injected every 4 hours.

• Courtesy of Ethicon Suture Laboratories Johnson & Johnson

line and the opening of the tube is set 3 or 4 mm below the surface of a level of fluid through which the air bubbles. The bottle is set on the floor next to the bed. If it is necessary to leave the needle in for more than a few hours it may be anchored obliquely against the chest wall with adhesive. Meantime the patient is given prophylactic chemotherapy with sulfonamide or penicillin (p. 3951).

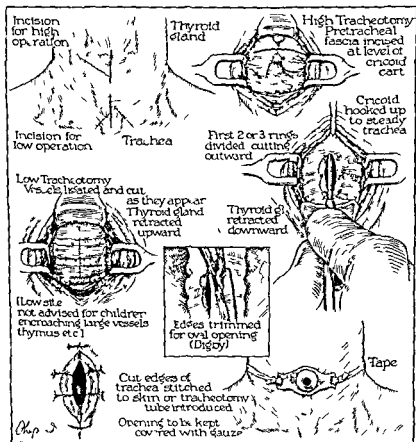


Fig. 1154.—Tracheotomy

Release of Blood that has Extravasated into Tissues of the Head and Neck—Massive concussion of the chest may result in the extravasation of blood into the fascial planes of the head and neck. In this region the veins possess no valves and an alarming accumulation of blood may occur with resultant cyanosis and threatened asphyxiation.

In the presence of serious symptoms releasing incisions are made through the deep cervical fascia. They are placed after adequate skin preparation parallel and anterior to the sternomastoids and are carried to a sufficient depth to relieve the trapped air or the blood clot.

Sealing of the Sucking Wound of the Chest—The sucking wound of

tuted immediately for the prevention and treatment of *shock* the control of *hemorrhage* and the correction of *exsanguination*

Relief of Tension Pneumothorax—A tension pneumothorax may result from an injury to the chest with or without penetration of the skin. The presence of the condition may be obvious from subcutaneous crepitation, chest pain and the physical signs or it may only be apparent later when

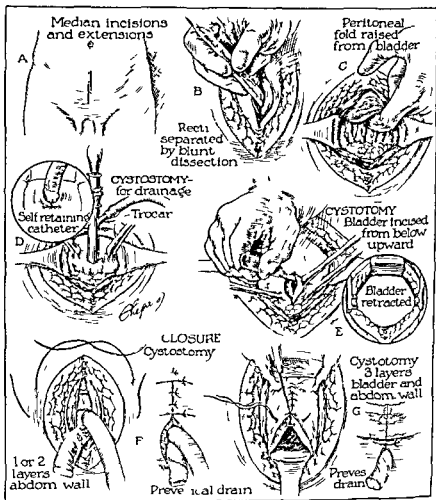


Fig 11.3—Suprapubic cystostomy and cystotomy

the progression of dyspnea and the displacement of the mediastinum become alarming. See *Differential Diagnosis of Pneumothorax* (p 2035)

The correction of the condition is accomplished after anesthetization of the skin of the chest wall. A small incision is made and a blunt 14 gauge needle is gently introduced into the pleural cavity. The needle is protected by a sterile cork so that it cannot possibly penetrate further than the surgeon desires. By a connecting piece the needle is attached to approximately 3 feet of sterile rubber tubing which may be obtained from an intravenous infusion set. The free end of the rubber tubing is placed in a bottle of

Courtesy of Ethicon Suture Laboratories Johnson & Johnson

saline and the opening of the tube is set 3 or 4 mm below the surface of a level of fluid through which the air bubbles. The bottle is set on the floor next to the bed. If it is necessary to leave the needle in for more than a few hours it may be anchored obliquely against the chest wall with adhesive. Meantime the patient is given prophylactic chemotherapy with sulfonamide or penicillin (p 3951)

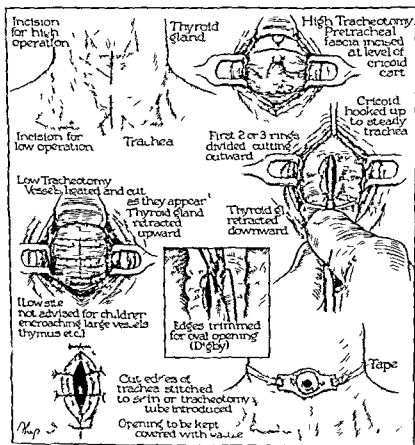


Fig 1154—Tracheotomy

Release of Blood that has Extravasated into Tissues of the Head and Neck—Massive concussion of the chest may result in the extravasation of blood into the fascial planes of the head and neck. In this region the veins possess no valves and an alarming accumulation of blood may occur with resultant cyanosis and threatened asphyxiation.

In the presence of serious symptoms releasing incisions are made through the deep cervical fascia. They are placed after adequate skin preparation parallel and anterior to the sternomastoids and are carried to a sufficient depth to relieve the trapped air or the blood clot.

Sealing of the Sucking Wound of the Chest—The "sucking wound of

the chest is sealed with an air tight dressing meantime the patient is given prophylactic chemotherapy with sulfonamide or penicillin or a combination of both

Confusion of the Heart and Cardiac Tamponade—See *The Circulatory System* (p 872)

Cystostomy for Retention of Urine—With injuries to the perineum and urethra it may be impossible to pass a catheter and a cystostomy is required

Under anesthesia the patient is placed on his back with feet and trunk moderately elevated. A suprapubic midline incision is made through skin subcutaneous tissues and the sheath of the rectus the pyramidalis if encountered is split laterally and retracted. With the bladder distended access to the space of Retzius is readily attained. The peritoneum is stripped from below upward by blunt dissection and the anterior wall of the bladder is exposed. It is seized with two Allis clamps and the urine is drained by the use of a side arm suction trochar. An indwelling mushroom catheter is inserted and sewn tightly into the bladder wall. Sulfonamide powder is applied locally and the skin is closed with interrupted sutures. Continuous suction is maintained on the catheter and fluids are given freely to insure a daily output of 1500 cc of urine. Prophylactic antibiotic treatment is started by intramuscular injections of 50 000 units of penicillin every 3 or 4 hours (Fig 1153 p 3956)

Tracheotomy—It is essential that the practitioner be prepared to perform tracheotomy in an acute emergency. The procedure may be required in severe laryngeal obstruction supraglottic edema or impaction of a foreign body (Fig 1154 p 3957)

- 1 Place the patient on his back with sandbags between the shoulders so that head and neck are extended and chin is exactly in the median line
- 2 Cleanse the skin of the neck and paint with 3½ per cent Tincture of Iodine
- 3 Induce local anesthesia by injection of 2 per cent procaine along the line extending from the notch of the thyroid cartilage to the manubrium. Continue the injection to the lateral portions of the neck so that a diamond is formed
- 4 Make a median line incision using the symphysis of the jaw and the manubrium as guides. The incision should extend from just below the thyroid cartilage to the suprasternal notch
- 5 Deepen the incision to expose the fascia between the infrahyoid muscles. Cut this fascia with a scissors and bring the trachea into view
- 6 Retract the isthmus of the thyroid gland downward or doubly ligate and incise if the trachea cannot be cleanly exposed without this procedure
- 7 Expose the upper four or five tracheal rings. Inject directly into the trachea with a hypodermic needle about 0.25 cc of 10 per cent cocaine with one drop of 1:1000 epinephrine
- 8 Introduce a hook into the upper tracheal ring and pull the trachea forward

- 9 Incise the third or fourth tracheal cartilages in the midline and insert a Trousseau dilator so that the tracheal lumen can be visualized
- 10 Trim the edges of tracheal incision to make an oval shaped opening
- 11 Fix tracheal edge by through and through sutures
- 12 Introduce a tracheotomy tube using the largest size that will comfortably fit the tracheal diameter
- 13 Hold tracheal tube in place by insertion of tape tied firmly around the neck
- 14 Loosely pack the wound with iodoform gauze
- 15 Introduce a skin suture at the upper angle
- 16 Have available a suction apparatus and a catheter to clear the air way and maintain constant nursing observation
- 17 Start intramuscular injections of 50 000 units of penicillin every 3 or 4 hours

LOCAL TREATMENT OF THE TRAUMATIC CONTAMINATED OR INFECTED WOUND

The surgical principles which underlie the treatment of the traumatic contaminated or infected wound are the following

- | | |
|---|---|
| 1 Control of bleeding | 5 Topical chemotherapy |
| 2 Anesthesia | 6 Incision and drainage for suppuration |
| 3 Wound cleansing and debridement | 7 Wound closure |
| 4 Measures aimed at the localisation of infection | 8 Restoration of function |

The Control of Bleeding

Whereas the remaining measures for treatment of the traumatic contaminated or infected wound may wait until the systemic disturbances

TABLE 21⁰—LOCAL MEASURES FOR CONTROL OF BLEEDING

Touquet

For bleeding in extremities loosen every 10 or 15 minutes to prevent circulatory difficulties

Local Pressure

Applied by the use of sterile gauze or a sponge forceps latter particularly useful in cavities such as the tonsillar bed and the vagina or rectum

Application of Vasocostrictor

Use epinephrine 1:1000 solution or powdered epinephrine diluting of secondary vasodilatation in painful area anesthetize first with 2 per cent diethane

Application of Coagulant

Use oxidized gauze topical thrombin or fibrin foam

Cauterization

Use a drop of trichloroacetic or chromic acid for a small bleeding point such as in the nose may use actual cautery after anesthesia

Packing

Particularly for cavities such as the nose vagina and rectum or the tonsillar fossa

Hemostasis

After local anesthesia tie a bleeding point or use a mass ligation with transfexion

(p 3952) have received attention the control of bleeding is the immediate concern of the practitioner. In Table 242 are tabulated the local measures which are available and which may be combined for more effective action.

Local Care of the Contaminated Wound

- 1 Cover with sterile gauze
- 2 Shave clean and prepare surrounding area

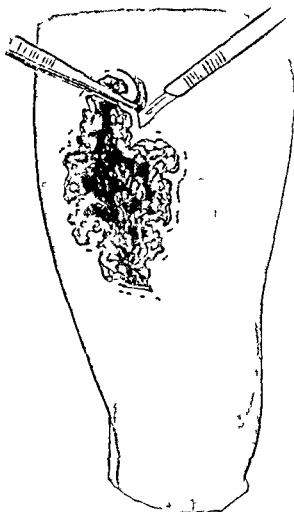


Fig 1155—Debridement technic. Margins of wound excised sparingly including underlying tissues to bone level.

- 3 Anesthetize with 2 per cent procaine pack or by infiltration field or nerve block
- 4 Secure bleeding points
- 5 Remove gross particles with forceps
- 6 Flush with saline solution retracting wound edges to get at corners and depths of wound avoid chemical antiseptics

- 7 With gloves scrub wound with soap on cotton pledget held in artery or dressing forceps
- 8 Remove soap with saline flush
- 9 Bubble in solution of hydrogen peroxide
- 10 Practice debridement if necessary trim off skin edges and remove traumatized fat fascia and muscle avoid injury to tendons nerves and larger vessels
- 11 Consider use of sulfanilamide powder (p. 98)

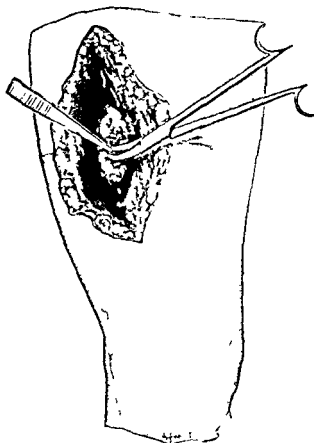


Fig. 1156—Debridement technic. Tufted surface removed; this is "epitachage."

- 12 Close by primary suture if possible; if in doubt leave in small drain for 24 hours
- 13 If wound is large use relaxing incision or pack for secondary suture
- 14 Start intramuscular injections of 50,000 units of penicillin every 3 or 4 hours

Local Care of the Infected Wound

The local care of the infected wound is determined by the nature of the inflammatory process. Different principles prevail in the presence of

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cellulitis lymphangitis and lymphadenitis thrombophlebitis erysipelas and suppuration

CELLULITIS LYMPHANGITIS AND LYMPHADENITIS

Systemic Treatment

- 1 Give sedatives and analgesics
- 2 If temperature is elevated or patient appears toxic insist on bed rest
- 3 Force fluids

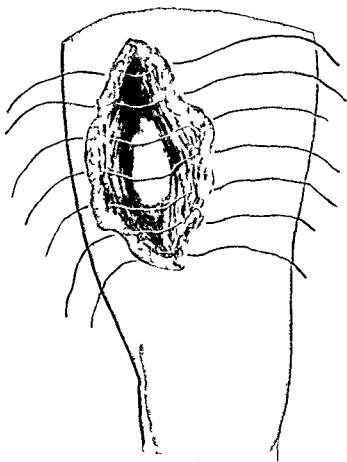


Fig 1157—Debridement technique Sutures placed but left untied (primo secondary or delayed suture) *

- 4 In *cellulitis* consider chemotherapy with sulfonamide or penicillin or both (p 3951)
- 5 In *lymphangitis* and *lymphadenitis* chemotherapy is imperative and must be started at once Use sulfonamide if ambulatory (p 88) and penicillin if injection therapy is feasible (p 106)

Local Treatment

- 1 Apply continuous warm wet dressings by open (p 3134) or closed (p 3134) methods

* Moorhead Clinical Traumatic Surgery

- 2 Protect adjacent
- 3 Immobilize
- 4 Consider roe
- 5 Avoid surgery

Systemic Treatment

- 1 Give sedatives
- 2 If temperature
- 3 Force fluids
- 4 Chemotherapy

Local Treatment

- 1 Prepare for surgery possible (in

Systemic Treatment

- 1 Give sedatives
- 2 If temperature rest
- 3 Force fluids
- 4 Start chemotherapy
- 5 Consider tetanus anaerobic

Local Treatment

- 1 Cover with sterile
- 2 Anesthetize by infiltration
- 3 Prepare skin in wound
- 4 Flush and cl
- 5 Incise skin
- 6 Trim and resect
- 7 Bubble in
- 8 Consider use
- 9 Pack or dress
- 10 Observe in
- 11 As soon as If necessary graft
- 12 Encourage
- 13 Protect u

TYPES OF T₁

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| | |
|-----------------------------------|------------------------------|
| Contusion Ecchymosis and Hematoma | Furuncle |
| Constriction | Carbuncle |
| Sprain | Finger Infection |
| Dislocation | Hand Infection |
| Fracture | Toe and Bunion Infection |
| Abrasion | Breast Abscess |
| Avulsion | Perianal and Crypt Abscesses |
| Incised Wound | Fistula in Ano |
| Lacerated Wound | Burns |
| Puncture Wound | Frostbite |
| Bite (Cat Dog Rat Snake Human) | Foreign Bodies |

CONTUSION ECCHYMOSIS OR HEMATOMA

Systemic Treatment

- 1 Analgesia
- 2 Seek injury to deep structures (p 3952)

Local Treatment

- 1 Apply pressure over cold compress or ice pack
- 2 Aspirate fluid blood in broad area

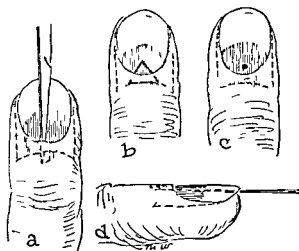


Fig 1158—Methods of decompression of a subungual hematoma a A sharp-pointed scalpel lifts the eponychium away from the base of the nail b A V shaped piece is excised from the base of the nail c A small hole is drilled through nail d A needle is inserted under the nail from the distal end

- 3 'Turn out clot in ear lobe to prevent cauliflower'
- 4 Release blood under nail bed in *subungual hematoma* to relieve pain
- 5 Pressure dressing over soft parts

CONSTRICTION

Systemic Treatment

- 1 Analgesia

Local Treatment

- 1 For finger ring induce digital block
- 2 Get ring cutter from jeweler or circular saw blade and remove

• Christopher Minor Surgery

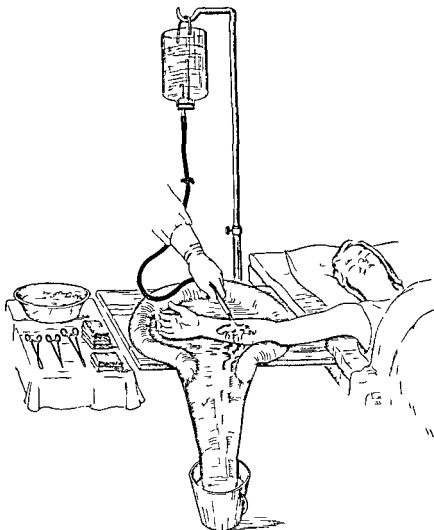


FIG. 1153.—Method of cleaning a fresh wound. For purposes of clarity the sterile towel which is placed under the arm is omitted so that the Kelly pad may be better seen. At the left is a small set up on a table consisting of sterile instruments, sterile pledgets of gauze and a solution of soap suds. The soap solution is made from white soap flakes in tepid or warm water and is agitated so that a real suds is formed. A pledget of gauze is first placed on the wound and the surrounding skin is thoroughly cleansed with the soap solution and washed with water. The pledget is then removed from the wound and the wound itself is then cleansed with the soap solution and washed with sterile water. This is done repeatedly. Finally a prolonged irrigation with sterile water is carried out. After this the Kelly pad is removed and the arm is redraped with sterile towels.

SPRAIN

Systemic Treatment

- 1 Analgesia. Repeat dose later when local anesthetic wears off
- 2 Seek evidence of deep injury

* Christopher, Minor Surgery

Local Treatment

- 1 Spray with ethyl chloride
- 2 Inject 2 per cent procaine into painful area
- 3 Apply elastic bandage and encourage movement
- 4 Get roentgenogram

DISLOCATION

See *Skeletal System* (p 2964)

FRACTURE

See *Skeletal System* (p 2982)

ABRASION**Systemic Treatment**

- 1 Analgesia

Local Treatment

- 1 Procaine pack or field block
- 2 Thorough cleansing and flushing
- 3 Debridement if necessary
- 4 No dressing if on face May paint with 3 per cent gentian violet
- 5 Elsewhere apply paraffin mesh gauze and dry dressing

AVULSION**Systemic Treatment**

- 1 Heavy sedation
- 2 Prevent or treat shock with hypodermics of opiate or demerol and 1 per cent neosynephrine
- 3 Prepare for plasma infusion if necessary
- 4 Initiate systemic chemotherapy with sulfonamide or penicillin

Local Treatment

- 1 Topical infiltration or block anesthesia If wound is extensive prepare for general anesthesia by intravenous pentothal or inhalation
- 2 Cleanse and debride wound
- 3 Secure hemostasis
- 4 Consider use of powdered sulfanilamide (p 99)
- 5 Replace flap after freshening edges
- 6 Suture loosely
- 7 If edges cannot be approximated try relaxing incisions (p 3931) or prepare for graft (p 3934)
- 8 Apply dry dressing
- 9 Observe in 24 hours Start intramuscular injections of 50 000 units penicillin every 3 or 4 hours

INCISED WOUND**Systemic Treatment**

- 1 Analgesia
- 2 Prevent or treat shock with injections of opiate or demerol and 1 per cent neosynephrine
- 3 Prepare for plasma infusion if necessary
- 4 Seek evidence of tendon nerve or deep injury (p 3952)

Local Treatment

- 1 Anesthesia by field or conduction block substitute general by intra venous pentothal or inhalation if necessary
- 2 Retract wound to ascertain injury to deep parts
- 3 Consider use of sulfonamide powder (p 99)
- 4 Close by primary suture
- 5 Apply dry dressing Start intramuscular injections of 50 000 units of penicillin every 3 or 4 hours
- 6 Observe in 24 hours

LACERATED WOUND

Systemic Treatment

- 1 Analgesia

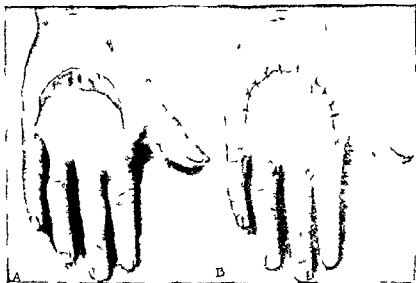


Fig 1160—A An avulsed flap of skin laid back in place unsutured but viable B An avulsed flap of skin sutured and doomed to necrosis as a result of anemias due to tension. In A a very slight scar results. In B skin grafting is often necessary.

- 2 Prevent or treat shock with injections of opiate or demerol and 1 per cent neosynephrine
- 3 Prepare for plasma infusion if necessary
- 4 Seek evidence of tendon nerve or deep injury (p 3952)
- 5 Start intramuscular injections of 50 000 units of penicillin every 3 or 4 hours
- 6 Consider tetanus gas gangrene antitoxin if wound is soiled or exposed to fecal contamination. Be sure to desensitize if there is a history of allergy or positive skin or conjunctival tests

Local Treatment

- 1 Anesthesia by field or conduction block substitute general by intra venous pentothal or inhalation if necessary

- 2 Debride thoroughly
- 3 Cleanse thoroughly
- 4 Retract to note injury to deep tissues
- 5 Consider use of sulfonamide powder (p 99)
- 6 Consider primary suture if wound is neither too deep too extensive nor too contaminated
- 7 If in doubt concerning number 6 (above) pack and prepare for secondary suture

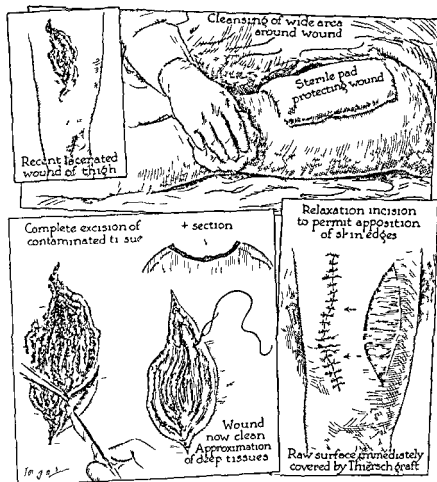


Fig 1161—Treatment of lacerated wound

- 8 Apply dry dressing
- 9 Observe in 24 hours

PUNCTURED WOUND

Systemic Therapy

- 1 Analgesia
- 2 Prevent or treat shock with injections of opiate or demerol and 1 per cent neosynephrine

Courtesy of Ethicon Suture Laboratories Johnson & Johnson

- 3 Prepare for plasma infusion if necessary
- 4 Seek evidence of tendon nerve or deep injury (p 3952)
- 5 Start intramuscular injections of 50 000 units of penicillin every 3 or 4 hours
- 6 Consider tetanus gas gangrene antitoxin if wound is soiled or exposed to fecal contamination Be sure to desensitize if there is a history of allergy or positive skin or conjunctival tests
- 7 Seek evidences of deep injury if penetration involved chest abdomen perineum or lumbar regions

Local Therapy

- 1 If wound is on face or extremities definitely contains no foreign body and was inflicted with a clean instrument apply dry dressing and observe
- 2 If foreign body has been introduced if instrument was unclean or if wound occurred through grossly contaminated area (perineum dirty hands or feet) anesthetize and treat as lacerated wound with attempted removal of foreign bodies (bullets clothing etc)

BITES (CAT OR DOG)

Systemic Therapy

- 1 Analgesia
- 2 Prevent or treat shock with injections of opiate or demerol and 1 per cent neosynephrine
- 3 Prepare for plasma infusion if necessary
- 4 Seek evidence of tendon nerve or deep injury (p 3952)
- 5 Start intramuscular injections of 50 000 units of penicillin every 3 or 4 hours
- 6 Consider tetanus gas gangrene antitoxin if wound is soiled or exposed to fecal contamination Be sure to desensitize if there is a history of allergy or positive skin or conjunctival tests
- 7 Seek evidences of deep injury if penetration involved chest abdomen perineum or lumbar regions
- 8 Apprehend animal and keep for observation for rabies (p 439)
- 9 If animal is normal after 14 days discontinue observation
- 10 If animal is rabid or cannot be located begin anti rabic vaccine in 15 dose series at daily intervals

Local Therapy

- 1 Anesthetize by local methods if on trunk or extremities Prepare for general anesthesia with intravenous pentothal or inhalation if bites are multiple or on face
- 2 Cauterize with fuming nitric acid even on face
- 3 Remove acid with paste of bicarbonate and water

BITE (RAT)

Systemic Therapy

- 1 Analgesia
- 2 Prevent or treat shock with injections of opiate or demerol and 1 per cent neosynephrine
- 3 Prepare for plasma infusion if necessary
- 4 Seek evidence of tendon nerve or deep injury (p 3952)

- 2 Debride thoroughly
- 3 Cleanse thoroughly
- 4 Retract to note injury to deep tissues
- 5 Consider use of sulfonamide powder (p 99)
- 6 Consider primary suture if wound is neither too deep too extensive nor too contaminated
- 7 If in doubt concerning number 6 (above) pack and prepare for secondary suture

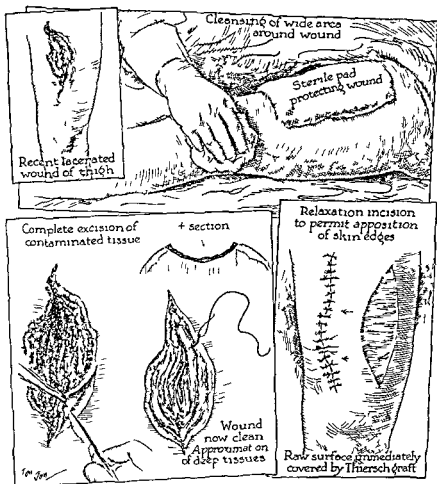


Fig 1161—Treatment of lacerated wound

- 8 Apply dry dressing
- 9 Observe in 24 hours

PUNCTURED WOUND

Systemic Therapy

- 1 Analgesia
- 2 Prevent or treat shock with injections of opiate or demerol and 1 per cent neosynephrine

Courtesy of Ethicon Suture Laboratories Johnson & Johnson

- 3 Irrigate with hydrogen peroxide
- 4 Pack open
- 5 Immobilize if on hand or knuckles in position of function

FURUNCLES

Systemic Treatment

- 1 Correct errors of general hygiene Clear glycosuria Reduce hyperglycemia Stop sweets in diet

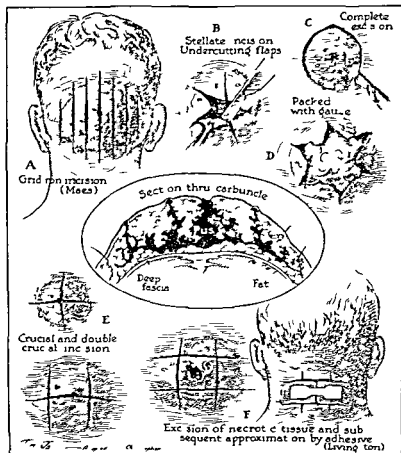


Fig 1162 ~Surgical treatment of carbuncle

- 2 Correct errors of local hygiene Substitute soap water and scrubbing brush for cosmetics Use lotions and avoid ointments
- 3 Insert drainage tube and carry through to lower quadrant and counened systemic complications Do not delay if furuncle is above upper lip or in external auditory canal
- 4 Try autogenous vaccine as prophylactic

Courtesy of Ethicon Suture Laboratories Johnson & Johnson

- 5 Start intramuscular injections of 50 000 units of penicillin every 3 or 4 hours
- 6 Consider tetanus gas gangrene antitoxin if wound is soiled or exposed to fecal contamination Be sure to desensitize if there is a history of allergy or positive skin or conjunctival tests
- 7 Seek evidences of deep injury if penetration involved chest abdomen perineum or lumbar regions

Local Therapy

- 1 If wound is on face or extremities and contains no visible foreign body apply dry dressing and observe
- 2 If wound occurred through grossly contaminated area anesthetize and treat as lacerated wound

BITE (SNAKE)

Systemic Therapy

- 1 Analgesia
- 2 Prevent or treat shock with injections of opiate or demerol and 1 per cent neosynephrine
- 3 Prepare for plasma infusion if necessary
- 4 If snake is identified as poisonous or is unidentified in area known to be inhabited by poisonous snakes give antivenin (p 83)

Local Therapy

- 1 Apply tourniquet
- 2 Anesthetize by field block
- 3 Make cruciate incision and aspirate
- 4 Apply dry dressing

BITE (HUMAN)

Systemic Therapy

- 1 Analgesia
- 2 Prevent or treat shock with injections of opiate or demerol and 1 per cent neosynephrine
- 3 Prepare for plasma infusion if necessary
- 4 Seek evidence of tendon nerve or deep injury (p 3953)
- 5 Start intramuscular injections of 50 000 units of penicillin every 2 hours
- 6 Consider tetanus gas gangrene antitoxin if wound is soiled or exposed to fecal contamination Be sure to desensitize if there is a history of allergy or positive skin or conjunctival tests
- 7 Seek evidences of deep injury if penetration involved chest abdomen perineum or lumbar regions
- 8 Get Wassermann on assualant and look for primary or secondary lesions especially in mouth
- 9 Take Wassermann and repeat weekly for 6 weeks

Local Therapy

- 1 If wound is on face or extremities and contains no visible foreign body apply dry dressing and observe
- 2 If wound occurred through grossly contaminated area anesthetize and treat as lacerated wound (p 3959)

Local Treatment

- 1 Avoid local trauma
- 2 Apply bulky wet and hot boric acid compresses
- 3 Use roentgen therapy if possible
- 4 Try to avoid incision except to liberate fluid pus and for removal of necrotic tissue
- 5 If necr. are open by gridiron or cruciate incision Pack and drain
- 6 Dress daily
- 7 When granulating approximate with flamed adhesive If necessary close defect by flaps or grafts

FINGER INFECTIONS**Definitions**

- Eponychia* and *Paronychia* Infections of tissues around the nail
Subungual Abscess Extension of infection under nail bed
Anterior Closed Space Infection (Felon) Infections of pulp of finger tip
Collar Button Abscess Superficial pocket connected by narrow sinus to larger and deeper pocket usually on palmar aspects of fingers or hand or in web spaces

Systemic Treatment

- 1 Correct errors of general hygiene Clear glycosuria Reduce hyperglycemia Stop sweets in diet
- 2 Start chemotherapy with sulfadiazine and/or penicillin without delay
- 3 Force fluids
- 4 Analgesia and sedation

Local Treatment

- 1 Apply heat by immersion in hot water
- 2 When localization is sharp anesthetize by finger block
- 3 Incise and drain as illustrated
- 4 Consider use of sulfonamide powder (p 99)
- 5 Apply bulky hot wet dressings
- 6 Inspect at 24 hours and remove drain if possible
- 7 If healing is delayed suspect osteomyelitis and get film

HAND INFECTIONS

Because of the grave sequels of hand infections the practitioner should treat these lesions only if specialist consultation is not available Under any circumstance operating room technic is required with an assistant to re tract general anesthesia is strongly suggested Systemic treatment is applied as for the carbuncle (p 3972)

The hands contain many wide open fascial spaces and bursae in which there are few barriers to the spread of infection Minor wounds especially puncture wounds involving these spaces may produce rapidly spreading infections involving the entire space

ANATOMIC REVIEW

The chief pathways for the spread of infections are the *tendon sheaths lumbrical canals* and *fascial spaces* The flexor tendon sheaths of the second, third fourth fingers extend from the distal interphalangeal joint of the respective fingers to the distal palmar flexion crease Those of the thumb and fifth fingers extend into the palm where they expand to form re-

Local Treatment

- 1 Avoid local trauma
- 2 Apply bulky wet and hot boric acid compresses
- 3 If above upper lip consider roentgen therapy
- 4 When clearly pointed open with phenol on toothpick or nick with scalpel

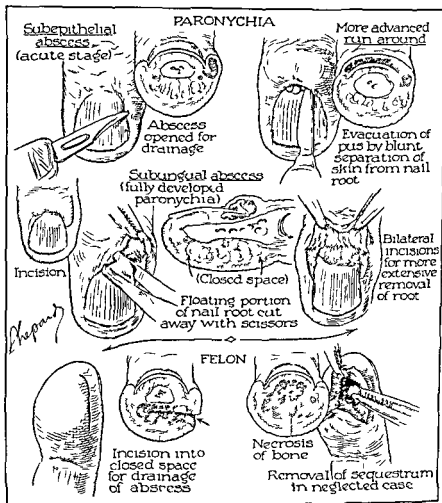


Fig 1163—Surgical treatment for paronychia and felon

- 5 Apply 5 per cent sulfathiazole ointment to neighboring tissue especially lds collar line (p 99)

CARBUNCLE

Systemic Treatment

- 1 Correct errors of general hygiene Clear glycosuria Reduce hyperglycemia Stop sweets in diet
- 2 Start chemotherapy with sulfadiazine and/or penicillin without delay
- 3 Force fluids
- 4 Analgesia and sedation

Courtesy of Ethicon Suture Laboratories Johnson & Johnson

The *hypothenar space* lies over the *hypothenar muscles*. It is well localized and infection in it is unlikely to spread.

The *subaponeurotic* and *subcutaneous spaces* are well localized spaces on the dorsum of the hand and are not particularly susceptible to spreading infection.

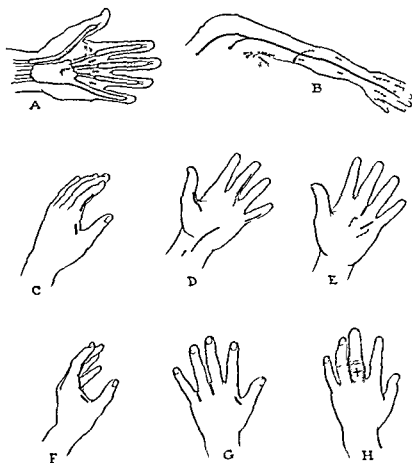


Fig 1165.—Incisions for the hands. *A* Relationship of palmar spaces to the tendon sheaths. *B* Lymphatic drainage system for fingers, hand, forearm, upper arm, shoulder and nodes at elbow and axilla. *C* Incisions for tenosynovitis of index finger. *D* Incisions for tenosynovitis of flexor tendon of thumb and fingers. *E* Incision for the drainage of mid palmar space infection. *F* Incision for drainage of infection of thenar space. *G* Accessory incisions for infection of web and thenar spaces. *H* Location of crucial incision for carbuncle of finger.

Infections of the tendon sheaths or fascial spaces of the hand may be the result of direct implantation or of spread from other sites by the routes mentioned above. After accurate diagnosis of the localization of the infection before classical fluctuation becomes manifest, a suitable incision is made. Delay causes irreparable damage to tendons and joints as well as spread to other fascial spaces.

Technic of Applying an Esmarch Bandage.—The Esmarch bandage

spectively the *radial* and *ulnar bursae*. The radial and ulnar bursae may communicate with each other thus permitting infection introduced into the first or fifth tendon sheath to spread into the other without involvement of the sheaths of the second third and fourth fingers. The bursae also extend beneath the annular ligament of the wrist into the forearm.

The *lumbical canals* extend from the dorsum of the base of each phalanx to the flexor tendon in the middle of the palm. They provide a route for the extension of pus from infections about the metacarpophalangeal joint to the palmar spaces.

The hand has in addition five *fascial spaces* which may become infected by extension from tendon sheaths and adjacent soft tissues or by direct implantation.

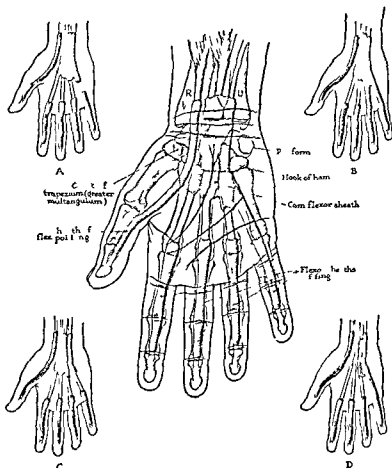


Fig 1164—Tendon sheaths of the palmar surface of the right hand shown in relation to skeleton and surface markings A B C and D represent variations

The *middle palmar space* lies deep to the tendons of the little ring and middle fingers and their accompanying vessels and nerves. It occupies the concavity of the palm. It may be infected from tenosynovitis of the third fourth or fifth finger through the corresponding lumbical canals from the ulnar bursa or from an osteomyelitis of the middle or ring metacarpals. Infection may spread from here to the thenar space or to the forearm.

The *thenar space* lies upon the adductor muscles of the thumb and is partially covered by the short muscles of the thenar eminence and by the flexor tendons of the index finger with its accompanying vessels and nerves. Infection may occur by direct implantation from a tenosynovitis of the index finger and thumb or by osteomyelitis of the thumb or index finger metacarpals. Infection may spread to the forearm or the mid palmar space.

acid solution. The gauze is held in place with a sterile bandage and this is covered with a rubber or other impervious sheet. The hand should be comfortably supported by a pillow. The entire bandage is kept warm by hot water bottles, a heating pad or lighted cradle. From time to time fresh solution is added through one corner of the dressing which is left open. These continuous wet fomentations are continued for 36 to 48 hours. At this time they may be discontinued so that the skin will not become unduly macerated. Also at this time drains are removed.

The extremity is treated by alternate periods of dry heat obtained by a lighted cradle and arm soaks in a sterile hot saline or boric acid solution. Persistent pyrexia after 48 or 72 hours indicates inadequacy of the incision and requires search for other foci of infection which may not have been noted at the original operation.

In 5 to 7 days drainage should be minimal and at that time gentle active motion can be instituted. This is best done while the arm is immersed in a hot soak and may be repeated two to three times a day for 10 to 15 minutes at each sitting.

In the intervals between the periods of active motion the hand is kept immobilized in the position of function using an appropriate metal or plaster splint. After the wound is healed active exercises and the splint are continued until function is normal. If the tendons have been irreparably damaged by late inadequate or improper incision purulent excretion will persist and under these circumstances it may be necessary to remove the tendon which now acts as a foreign body.

Infected Ingrown Toenail.—The commonest infection of the toes is the infected ingrown toenail. This usually consists of an infected granuloma of the eponychium resulting from continued irritation by the edge of the nail. It can be avoided if the patient cuts the toenails horizontally. Occasionally mild inflammations are aborted by hot foot soaks, the elimination of pressure by using a cut out shoe and elevation of the corner of the nail with a moist cotton wick.

If this therapy does not suffice the entire inner border of the nail is removed with a diagonal incision so made as to leave no sharp edge. Wet dressings are used for a few days.

While the nail is growing out a cotton wick is placed beneath the nail border. It is changed every two days. It protects the bed from pressure as the nail grows out. If treatment fails or the lesion recurs the more radical operation is necessary (p 3944).

Infected Bunions.—Infected bunions are treated with hot wet dressings. If conservative treatment is not effective the bunion is incised and drained. The incision should be placed so that it will not overlie a pressure point.

After the incision is healed a more radical procedure with excision of the causal factor should be performed.

BREAST ABSCESS

The breast abscess is most often encountered during the period of lactation. If chemotherapy with the sulfonamides seems indicated the practitioner must be mindful of the fact that the chemotherapeutic agent is excreted in the milk and may cause toxic manifestations in the nursing

employed before dealing with hand infections. It is a constricting rubber bandage which is begun at the distal portion of the extremity and wound with successive turns proximally. Each successive turn should overlap the preceding by about $\frac{1}{4}$ inch. Sufficient pressure should be applied to cut off the arterial circulation.

After the entire bandage is placed it is unwound beginning at the distal portion by pulling out each successive turn. The last three or four turns are not disturbed and the end of the bandage is tied snugly to hold it in place. Possible damage to underlying tissues is avoided if the portion of the extremity on which the bandage is to remain is first covered with a folded towel and the bandage is applied over this.

Tendon Sheath Infections—Tendon sheath infections give a characteristic clinical picture. The finger is held rigidly semi flexed and any attempt at extension of the finger gives exquisite pain localized maximally at the proximal limit of the sheath. Tenderness is present over the entire course of the tendon sheath.

Incisions are made over the entire course of the sheath. They are to be made on the lateral or dorsolateral aspects of the finger to avoid subsequent restricting scars and damage to the digital vessels or nerves. The sheaths are incised for their entire length merely preserving the thickenings over the interphalangeal joints lest the tendons subsequently prolapse. Incisions are best made bilaterally but through and through drain should *not* be used. The tendon itself should not be disturbed or traumatized.

Mid Palmar Space Infections—In mid palmar space infection there is a history of appropriate trauma or preceding infection followed by pain and fulness of the palm and swelling of the entire hand. There may be marked edema of the dorsum overshadowing the causal lesion of the palm. Tenderness is elicited over the anatomical location of the space.

Incision is made in the distal flexion crease beginning over the middle metacarpal and extending medially as far as the fourth metacarpal. The incision curves proximally to the proximal flexion crease. The tendons of the third and fourth fingers are carefully retracted and the palmar space is opened.

Through and through drainage to the dorsum is avoided.

Thenar Space Infections—With thenar space infection inspection reveals fulness of the lateral half of the palm and there is tenderness over the thenar eminence.

Hypothenar and Dorsal Space Infections—Incisions in the hypothenar and dorsal spaces are made over the fluctuant area. Infections in these areas remain localized and give the classical signs of suppuration.

Treatment After Incision—After incision subsequent care is extremely important. Dressings are made under strict aseptic precautions. Sterile gloves are worn to avoid secondary contamination.

Immediately after the operation the hand is enveloped in a dried fluff gauze bandage. This is so applied as to maintain the hand in a position of function that is slight dorsiflexion at the wrist and semiflexion of the fingers with apposition of the thumb to the fingers. This dry dressing is retained for from 6 to 12 hours until all bleeding has stopped. It is then replaced with a voluminous moist wet dressing. The hand and forearm are covered with large amounts of fluff gauze moistened with warm boric

PERIANAL AND CRYPT ABSCESSSES

The *perianal abscess* presents as a fluctuant tender perianal mass. A *crypt abscess* or *ischio rectal abscess* can be detected as a tender boggy mass on rectal examination.

Perianal abscesses should be incised by a radial incision placed near the anus so that the resultant fistula in ano will be as short as possible. Infil-

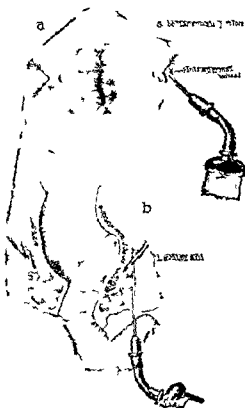


FIG. 1167.—Local anesthesia for minor surgery of the rectum. *a*, Lateral intradermal wheals made by injection of 0.5 per cent novocain under the papillary layer of the skin. Arrows indicate direction of needles put through these wheals to make a circular subcutaneous injection. *b*, Left index finger looks around the internal sphincter muscle. Long needle passing through an intradermal wheal through the peripheral fibers of the external sphincter muscle to the level of the levator ani muscle. The injections are carried out around the whole circumference of the rectum.

tration of the overlying skin with procaine provides sufficient anesthesia for small lesions. Larger ones require general or low spinal anesthesia. After incision the cavity should be packed. The packing is removed in 48 hours and slightly repacked. Following the first bowel movement hot sitz baths should be instituted two to three times a day and the patient should be instructed to cleanse the perianal skin carefully following each evacuation. The resultant fistula can be treated in about three weeks.

David V. C. Surgery of the Rectum and Anus in Nelson's Loose Leaf Surgery
New York: Thomas Nelson & Sons.

Systemic Treatment

- 1 Discontinue nursing
- 2 Start chemotherapy with penicillin if there are constitutional manifestations or the patient seems debilitated
- 3 Give liberal doses of analgesics and sedatives to overcome restlessness and sleeplessness
- 4 Force fluids
- 5 Consider the administration of androgen to inhibit lactation and dry the breasts

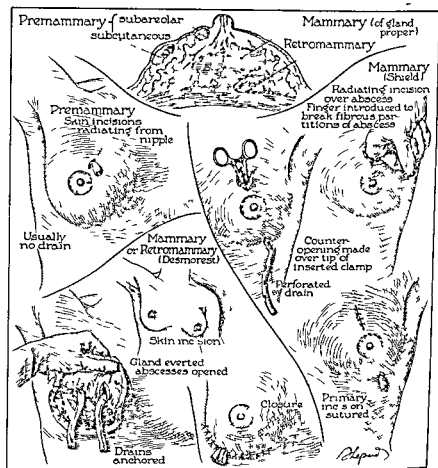


Fig 1166—Incision of breast

Local Treatment

- 1 Use bulky hot wet compresses until localization is sharp
- 2 When indicated give general anesthesia by intravenous or inhalation technic and make radial incision as indicated
- 3 Initiate chemotherapy with sulfathiazole and/or penicillin for threat
- 4 Suture the primary incision and drain through counter incision
- 5 Inspect in 24 hours and remove drain if possible at the time

* Courtesy of Ethicon Suture Laboratories Johnson & Johnson

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David, V. C. *Surgery of the Rectum and Anus in Nelson's Loose Leaf Surgery*. New York, Thomas Nelson & Sons.

Crypt abscesses can be visualized with an appropriate anoscope. Then a hooked probe can be inserted into the crypt and the mucosa overlying the probe should be incised to provide free drainage. The patient can then take hot sitz baths and hot rectal irrigations two to three times a day for a few days. Prompt subsidence of symptoms is to be expected.

FISTULA IN ANO

A *perianal discharge* usually indicates the presence of a fistula in ano. These lesions are secondary to perianal abscesses. Their treatment requires complete exposure of the tract from the external opening to the internal opening with the excision of the overhanging edges so as to create the flat wound. The operation can be performed either under low spinal or general anesthesia. Small fistulas can be treated easily by passing a probe

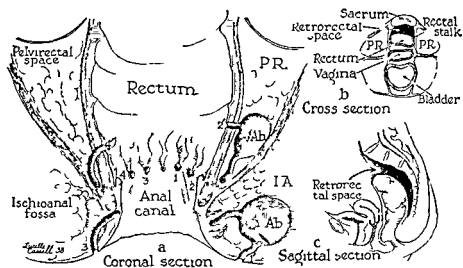


Fig. 1168—A diagrammatic representation of anal fistulas and of surgical spaces. *PR* indicates pelvirectal space. *IA* ischioanal space or fossa. *Ab* abscess. 1 2 3 4 fistulous tracts. 1 2 3 4 secondary openings.

from the external opening through to the internal opening. This probing is facilitated if the left index finger of the operator is first placed in the rectum. With the probe in place one can estimate the depth of the fistula. If the fistula is entirely superficial to the sphincter or at most lies deep to only the subcutaneous portion of the external sphincter, one need have no fear of subsequent incontinence after dividing all tissues overlying the probe. These can be divided. The fistulous tract itself may be either excised or thoroughly curetted and the resultant wound packed with iodoform gauze. The packing should not be left in place for longer than four or five days and then the patient should take hot sitz baths and observe the usual cleanliness as after any rectal operation. Such wounds should heal in about ten days.

Deeper fistulas are perhaps better treated by a multiple stage operation. At the initial operation the entire tract except for that portion which goes beneath the sphincter is completely opened and packed. A heavy silk

ligature is placed through the remaining portion of the fistula and tied loosely. The external wound is allowed to granulate in and heal and after two to four weeks the portion included in the ligature is incised under local anesthesia. With such a two stage procedure incontinence never occurs.

BURNS

The treatment of burns has undergone revolutionary change since the time of the Coconut Grove Night Club fire in Boston. The experiences in this catastrophe and in fires since that time have served to lay down these fundamental therapeutic principles.

The burn must be treated as a *surgical wound* under strictly aseptic conditions.

The *prime objective* of the surgeon is to convert a dirty and contaminated wound into one that is relatively clean. To accomplish this anesthesia is required; shock must be prevented or treated if present and professional assistance is essential for the successful accomplishment of debridement (p. 3960).

In making applications to the wound the *irritants* such as tannic acid and triple dye are avoided since they add to the tissue insult and delay healing.

Only *sterile ointments* are applied to the external surface and for this purpose petrolatum, boric acid and 5 per cent sulfadiazine are best utilized. The more irritant preparations such as the picric acid ointments may cause damage or produce manifestations of idiosyncrasy.

When the wound has been properly treated a *pressure bandage* is applied and dressings are not disturbed for approximately ten days unless some urgent indication arises. Frequent dressings produce trauma; they delay tissue healing and invite contamination and infection.

FIRST DEGREE BURNS: SIMPLE HYPEREMIA

Systemic Treatment

1. Analgesia and sedation

Local Treatment

1. Apply moist dressing of milk or saline solution
2. Dress with sterile petrolatum

SECOND DEGREE BURNS

Systemic Treatment

1. Analgesia and sedation
2. Prevent or treat shock with morphine 15 mg ($\frac{3}{4}$ grain) or demerol 50 mg ($\frac{1}{4}$ grain) and 1 cc of 1 per cent neosynephrine
3. Prepare for oral or intravenous use of $\frac{1}{6}$ molar sodium lactate or plasma infusion

Local Treatment

1. Insist on use of mask, gown and gloves with operating room technic
2. Prepare for local or general anesthesia
3. Clean, debride and irrigate
4. Cover with fine meshed sterile gauze impregnated with petrolatum, boric acid or 5 per cent sulfadiazine. Over this place several thick

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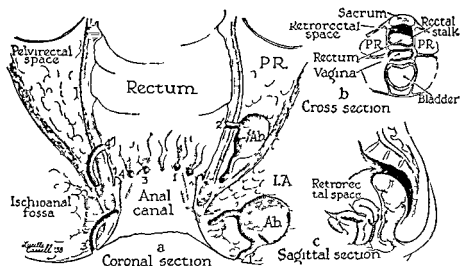


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- 2 Prevent or treat shock with morphine 15 mg ($\frac{1}{4}$ grain) or demerol 50 mg ($\frac{3}{4}$ grain) and 1 cc of 1 per cent neosynephrine
- 3 Prepare for oral or intravenous use of $\frac{1}{6}$ molar sodium lactate or plasma infusion

Local Treatment

- 1 Insist on use of mask, gown and gloves with operating room technic
- 2 Prepare for local or general anesthesia
- 3 Clean, debride and irrigate
- 4 Cover with fine meshed sterile gauze impregnated with petrolatum, boric acid or 5 per cent sulfadiazine. Over this place several thick

nesses of dry sterile gauze a gauze fluff and an abundance of mechanics waste or sea sponge

- 5 Apply mild pressure with stockinette for head and face elastic bandage for trunk and plaster of paris for extremities Extremity dressings must extend from tips of fingers or toes to 4 inches beyond burn Must be smooth and not constrictive
- 6 Leave dressings alone for 6 to 8 days to encourage epithelial regeneration
- 7 On removal of dressing cleanse with saline and peroxide
- 8 On 10th to 12th day prepare for graft if necessary

THIRD DEGREE BURNS

Systemic Treatment

- 1 Analgesia and sedation
- 2 Prevent or treat shock with morphine 15 mg ($\frac{1}{4}$ grain) or demerol 50 mg ($\frac{3}{4}$ grain) and 1 cc of 1 per cent neosynephrine
- 3 Prepare for oral or intravenous use of $\frac{1}{6}$ molar sodium lactate or plasma infusion
- 4 If mouth is involved use half strength peroxide as wash
- 5 If cornea is involved instill castor oil three times daily and apply wet compresses of boric acid
- 6 If larynx and trachea involved prepare for tracheotomy (p 3957) if required
- 7 Initiate chemotherapy with penicillin

Local Treatment

- 1 Insist on use of mask gown and gloves with operating room technic
- 2 Prepare for local or general anesthesia
- 3 Clean debride and irrigate
- 4 Cover with fine meshed sterile gauze impregnated with petrolatum boric acid or 5 per cent sulfadiazine Over this place several thicknesses of dry sterile gauze a gauze fluff and an abundance of mechanics waste or sea sponge
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- 7 On removal of dressing cleanse with saline and peroxide
- 8 On 10th to 12th day prepare for graft if necessary
- 9 Place on Bradford frame if necessary

FROST BITE

Systemic Treatment

- 1 Remove all clothing
- 2 Place in warm but not hot blankets
- 3 Try to raise temperature 2° F every half hour but no quicker
- 4 Avoid hot water bottles and pads
- 5 Give drink of whiskey every hour for 2 or 3 doses

- 6 Consider use of anticoagulants especially deposits of heparin by Loewe method (p 1050)

Local Treatment

- 1 Place extremities in iced saline for 30 minutes
- 2 Increase temperature of saline to 40° F for second half hour
- 3 Increase temperature of saline to room temperature (70° F) for third half hour
- 4 Apply packs of cold saline or milk to nose ears and genitalia
- 5 Cover skin lesions with boric acid ointment but avoid dressings
- 6 Encourage active motion frequently
- 7 Treat as second degree burn after 3 to 8 days

FOREIGN BODIES

Splinters—Splinters are the most common foreign bodies responsible for injury. They usually lie superficially, are readily seen and easily removed. If the splinter cannot be located by direct vision, *transillumination* (p 3633) makes the foreign body stand out in bold relief. This is a particularly valuable adjunct in the localization of splinters in the fingers and toes. Subungual splinters are readily recognized in this way.

The wound of entrance may have to be enlarged slightly so that the end of the splinter can be grasped with a fine forceps and then withdrawn. Occasionally only part of the splinter is removed in this way. The fragment lies so deeply that it is overlooked. If a patient gives a history of removal of a splinter followed by suppuration and a persisting draining sinus, there is strong likelihood of a retained portion of the splinter.

Removal of retained foreign bodies is quite simple. Under general block or local infiltration anesthesia the tract can be followed. A blunt pointed grooved director or probe is gently inserted in the tract. The tissues over the director are incised. The splinter is then identified and withdrawn with a fine forceps.

Subungual splinters are quite common and very painful. By the time the patient comes to the physician the splinter has been broken off flush with the nailbed so that it cannot be secured. A wedged shaped portion of the nail overlying the splinter is removed. The end then can usually be grasped with a fine forceps and the splinter withdrawn.

Foreign Bodies in Wounds—The removal from wounds of foreign bodies has been previously described (p 3965).

Following incision and drainage of abscesses, chronic drainage sinuses should be suspected of containing a foreign body. The tract is explored under general block or local infiltration anesthesia. A probe is placed in the tract and the overlying tissues are incised. Good light, careful hemostasis and retractors are essential. The foreign body may be felt before it is seen in the depth of the tract. It can be gently withdrawn with a forceps.

Foreign Bodies in Conjunctival Sac—Foreign bodies in the conjunctival sac are very common. A turned in eyelash (trichiasis) may give all of the symptoms of a foreign body. Most foreign bodies are located on the upper lid near its edge. The conjunctival sac should be explored in its entirety. The lower lid is depressed and inspected with the patient gazing upward.

The upper lid is then everted while the patient looks downward. An applicator is placed at the upper border of the upper lid. The lashes are pulled out and up while the applicator is pushed down and forward. Foreign bodies are readily detected near the free border of the upper lid. They can be removed with a moist cotton applicator.

If the foreign body is not found, the corner should be inspected carefully. Direct light will often obscure the foreign body. By indirect light it may appear as a tiny black speck with or without a surrounding narrow zone of corneal clouding. The corneal clouding may be white or if the particle is iron or steel it will be brown. Corneal foreign bodies should not be removed without first anesthetizing the corner with one or two drops of holocaine or butyn placed into the conjunctival sac. The particle if it is superficial can be removed with a tiny moist applicator. The patient should preferably be lying down and should fix his eyes by looking at a spot on the ceiling. Such particles can also be removed with a not too sharp eye spud or with the side of the bevel of a 19 gauge needle.

Cases with deep foreign bodies or a rust ring are best referred to an ophthalmologist. After removing a corneal foreign body it is wise to instill a small amount of 2 per cent boric acid ophthalmic ointment and one to two drops of homatropine (if the patient is less than 40 years of age) into the conjunctival sac and then bandaging the eye snugly for twenty-four hours.

Technic of Eye Bandage—Several layers of fluff gauze are placed over the closed upper lid forming a small mound. They are held in place by a layer of adhesive running from the mid point of the forehead to the cheek. Two more strips of 1 inch adhesive are then applied on each side of the central strip giving firm pressure.

Foreign Bodies in Ear—Foreign bodies in the external ear are removed by washing with an ear syringe. A large insect can first be killed by instilling a few drops of ether or chloroform. Ear lavage is prohibited in patients with drum perforations.

Foreign Bodies in Nose—Foreign bodies in the nose are removed by a nasal forceps or by suction. The nasal mucosa is shrunk with epinephrine or neosynephrine and anesthetized if necessary before instrumentation.

Foreign Bodies in Throat—Fishbones are the most common foreign bodies found in the throat. They usually become lodged in the tonsillar fossae or the base of the tongue. The patient usually localizes the foreign body quite accurately. The bone can be visualized under good light with or without the aid of laryngeal mirrors. Often only a tiny portion of the bone protrudes and the search must be quite careful and methodical. Once the foreign body is located it is readily grasped in a curved hemostat and can be withdrawn. If the foreign body cannot be located a lateral x ray of the neck should be taken to identify it.

Foreign Bodies in Esophagus—Foreign bodies in the esophagus give an immediate sensation of something being stuck either in the throat or the chest. The patient usually localizes the level of the lesion quite well. With such a history an x ray of the neck and chest both postero-anteriorly and laterally and esophagoscopy are essential.

Foreign Bodies in Respiratory Passages—Foreign bodies in the trachea and bronchi usually give paroxysms of coughing. They frequently may be

detected radiologically but may be radiotranslucent. Any patient who gives a history of having choked on something followed by a paroxysm of coughing and has persistent symptoms should be subjected to bronchoscopy.

Foreign Bodies in Digestive Tract—Foreign bodies in the gastro intestinal tract if they are radio opaque are readily located by a flat plate of the abdomen. Most of them will pass spontaneously. The patient is observed carefully and is cautioned to report any abdominal pain. He should be given a low residue diet. Repeated x rays and fluoroscopies permit one to follow the course of the foreign body but are not essential for treatment. If there are symptoms of peritoneal irritation (abdominal pain, tenderness, nausea, vomiting or abdominal rigidity) a laparotomy is mandatory.

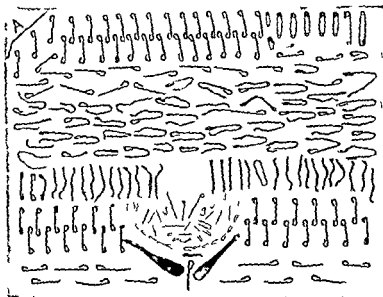


Fig. 1169.—204 articles swallowed by a mentally deranged patient. Many of these are sections of bed springs; included also are safety pins, spoon handles, pins and glass. Operation by Dr. Monteith of Nyack, N. Y.

Foreign Bodies in Rectum—All varieties of foreign bodies have been found in the rectum. Occasionally they can be felt manually and can be removed in that way. Otherwise by means of the sigmoidoscope the mucosa is inspected for evidence of perforation. Early perforations should be treated by laparotomy and suture. Delayed perforations are treated as other forms of peritonitis.

One type of rectal foreign body that requires special consideration is a *fecal impaction*. It occurs in a markedly constipated individual in elderly debilitated patients who are confined in bed for prolonged periods and occasionally in postoperative patients. Often one of the symptoms is a paradoxical diarrhea due to leakage of fluid feces around the impaction. The impaction is readily detected on digital examination and may be

The upper lid is then everted while the patient looks downward. An applicator is placed at the upper border of the upper lid. The lashes are pulled out and up while the applicator is pushed down and forward. Foreign bodies are readily detected near the free border of the upper lid. They can be removed with a moist cotton applicator.

If the foreign body is not found the cornea should be inspected carefully. Direct light will often obscure the foreign body. By indirect light it may appear as a tiny black speck with or without a surrounding narrow zone of corneal clouding. The corneal clouding may be white or if the particle is iron or steel it will be brown. Corneal foreign bodies should not be removed without first anesthetizing the cornea with one or two drops of holocaine or butyn placed into the conjunctival sac. The particle if it is superficial can be removed with a tiny moist applicator. The patient should preferably be lying down and should fix his eyes by looking at a spot on the ceiling. Such particles can also be removed with a not too sharp eye spud or with the side of the bevel of a 19 gauge needle.

Cases with deep foreign bodies or a rust ring are best referred to an ophthalmologist. After removing a corneal foreign body it is wise to instill a small amount of 2 per cent boric acid ophthalmic ointment and one to two drops of homatropine (if the patient is less than 40 years of age) into the conjunctival sac and then bandaging the eye snugly for twenty-four hours.

Technic of Eye Bandage—Several layers of fluff gauze are placed over the closed upper lid forming a small mound. They are held in place by a layer of adhesive running from the mid point of the forehead to the cheek. Two more strips of 1 inch adhesive are then applied on each side of the central strip giving firm pressure.

Foreign Bodies in Ear—Foreign bodies in the external ear are removed by washing with an ear syringe. A large insect can first be killed by instilling a few drops of ether or chloroform. Ear lavage is prohibited in patients with drum perforations.

Foreign Bodies in Nose—Foreign bodies in the nose are removed by a nasal forceps or by suction. The nasal mucosa is shrunk with epinephrine or neosynephrine and anesthetized if necessary before instrumentation.

Foreign Bodies in Throat—Fishbones are the most common foreign bodies found in the throat. They usually become lodged in the tonsils, the tonsillar fossae or the base of the tongue. The patient usually localizes the foreign body quite accurately. The bone can be visualized under good light with or without the aid of laryngeal mirror. Often only a tiny portion of the bone protrudes and the search must be quite careful and methodical. Once the foreign body is located it is readily grasped in a curved hemostat and can be withdrawn. If the foreign body cannot be located a lateral x ray of the neck should be taken to identify it.

Foreign Bodies in Esophagus—Foreign bodies in the esophagus give an immediate sensation of something being stuck either in the throat or the chest. The patient usually localizes the level of the lesion quite well. With such a history x ray of the neck and chest both postero-anteriorly and laterally and esophagoscopy are essential.

Foreign Bodies in Respiratory Passages—Foreign bodies in the trachea and bronchi usually give paroxysms of coughing. They frequently may be

If the defect is *arterial* pressure must be avoided. If the defect is *venous* properly applied compression to the entire limb will frequently improve the circulation by preventing venous stasis.

If an *arterial defect* is present smoking is prohibited and various *vaso dilator drugs* can be administered. Of these alcohol is perhaps the most useful. A drink of whiskey will often control the pain accompanying ulceration in thrombo angitis obliterans when as much as 30 to 60 mg ($\frac{1}{4}$ to 1 grain) of morphine will be insufficient. One half to one ounce of whiskey can be given from 3 to 4 times a day. Capsules containing 30 mg ($\frac{1}{2}$ grain) papaverine hydrochloride and 0.5 gm ($\frac{7}{10}$ grains) aminophylline or theobromine sodium salicylate can also be administered.

Various *mechanical measures* increase the peripheral circulation if the lesion is on the extremity. Perhaps the best are the *Buerger's exercises*. The patient sits on the side of the bed with his legs dangling for one minute or until marked rubor develops. He then lies down on the bed and

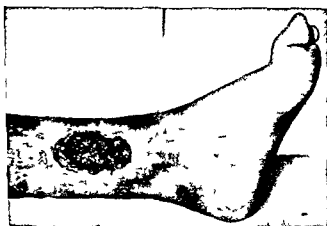


Fig 1170—Varicose ulcer

elevates the leg at an angle of 30 to 40 degrees placing it on some support if necessary for 2 minutes or longer until it blanches. Following this the legs are placed on the bed and the patient remains horizontal for three minutes. These cycles can be repeated about 5 to 10 times three times a day.

Mechanical appliances may be used to produce essentially the same effect as the Buerger's exercises. They are hardly more efficient but do not require the active cooperation of the patient. These are the *intermittent venous occlusion apparatus*, the *suction pressure boot* and the *oscillating bed*.

In thrombo angitis obliterans *hypertonic saline solution* may be given intravenously to increase the blood volume and decrease blood viscosity. Three hundred cubic centimeters of 5 per cent saline are administered on alternate days. This procedure is usually followed by a generalized vaso dilatation with rather considerable elevation of skin temperature which persists for several hours.

partially broken up manually This should be followed by an enema of 4 ounces of hydrogen peroxide and 8 ounces of water retained as long as possible The peroxide enema is advantageously followed by a colon irrigation or enema consisting of glycerin 80 magnesium sulfate 80 and soap suds to make 240 0 The entire process may have to be repeated two or three times before the impaction is completely removed

WOUND COMPLICATIONS

Any wound may become ulcerated or gangrenous at times the necrosis of tissue is due to the nature of the invading organism as occurs in gas gangrene (p 300), anthrax (p 292) the mycoses (p 489) tularemia (p 323) syphilis (p 331) rat bite fever (p 363) and tuberculosis (p 252) Only by smears and cultures taken from the wound can the exact diagnosis be established

Ulceration and gangrene may also result from local disturbances irrespective of the nature of the bacterial invader This situation prevails with venous varicosities when chemical antiseptics are used in overzealous excess as the result of tight bandaging from trauma to the local vasculature from malignancy or the use of a mass ligature Finally death of tissue is associated with systemic disability such as is encountered in uncontrolled diabetes mellitus (p 1246), generalized arteriosclerosis (p 981) thromboangitis obliterans (p 1029) cachexia (p 19) and malnutrition profound anemia (p 1055) or the blood dyscrasias (p 1096)

ULCERATION

An ulcer is an indolent inflammatory lesion in which there is a loss of substance of the skin and deeper tissues leaving a shallow defect with heaped up edges The base and walls of the ulcer are formed by granulation tissue covered by variable amounts of exudate There is usually present a mixture of several mildly pathogenic organisms which are essentially surface contaminants They are responsible for the local inflammatory reaction which commonly is present Occasionally they invade the organism giving rise to more specific lesions such as *cellulitis* or *erysipelas*

Treatment—The treatment of the ulcer consists in the main of measures to avoid and circumvent the precipitating factors Specific ulcerations require specific therapy In doubtful cases a Wassermann test is done to rule out syphilis and a biopsy is performed to exclude malignancy

The elimination of prolonged pressure requires careful nursing care The patient must be turned every 15 to 30 minutes first on the back then on the abdomen and then on either side Pressure over the back can be controlled more easily by placing the patient on an air mattress or water mattress which decreases the pressure over specific bony prominences These can also be protected by cushions cut in the shape of a doughnut with a central hole over the bony prominence It is well to place a cradle over the legs to support the bed clothes and prevent them from resting on the leg

Since the most frequent defect is circulatory the major therapeutic measures are directed toward promoting normal circulation These will differ depending on whether the defect is primarily arterial or venous

To apply the dressing the leg is elevated for about one half hour and then painted thoroughly with warm liquid paste. Over this a gauze bandage is applied from toes to knees over both the skin and ulcer. A second layer of paste is applied over the bandage and this is followed by another layer of bandage. In all three to four alternating layers of paste and gauze are applied. Such a boot may be left on for several weeks. Already prepared bandages impregnated with zinc oxide may be obtained from surgical supply houses. These are more convenient to use and are as efficient as the Unna's paste boot.

In addition to measures aimed at the treatment of the vascular defect (p 1030) the *base of the ulcer* requires attention. The *dry and clean* ulcer need only be protected by a dry sterile dressing. Ointments containing cod liver oil with zinc oxide (desitin ointment) are helpful.

If there is *secondary infection* with much moist sloughing tissue the ulcer is treated as any other infected wound. Great care is exercised to avoid maceration of the adjoining skin which can then easily break down and also become infected.

When there is *peripheral arterial disease* the moist dressings should be intermittent alternating with periods during which the lesion is covered merely by a dry sterile dressing so that the skin may dry.

The dressing is performed with strict aseptic precautions. The healthy skin surrounding the lesion is washed with soap and water using sterile cotton pledgets rinsed with sterile water and finally with 70 per cent alcohol which will aid in keeping it dry. Any mildly antiseptic solution such as azochloramide in oil 70 per cent alcohol or a saturated solution of boric acid may be applied to the ulcerated area itself. This can be done as a daily short soak in a warm solution followed by drying of the skin and ulcerated area. Later there may be applied a dry dressing or a moist dressing consisting of either 70 per cent alcohol or a mixture of equal parts of 70 per cent alcohol and saturated boric acid solution. These solutions evaporate in a few hours so that the dressing remains dry for the remainder of the day providing alternate moist and dry dressing.

Once the granulations at the base of the ulcer have become clean steady healing is to be anticipated if the local circulation is adequate. When mechanical appliances are used to produce reactive hyperemia additional damage must be prevented. No form of therapy that causes increased pain should be continued.

Large necrotic ulcerating lesions on the trunk unassociated with occlusive arterial disease (the so called *bed sores*) can be treated somewhat more radically. They can be irrigated continuously with Dakin's solution or with azochloramide without fear of macerating the skin. Grossly sloughing tissue can be excised without waiting for spontaneous separation. No anesthetic is needed since the tissues are dead and anesthetic. Healthy tissue must not be invaded. No bleeding should be caused by the excision.

Following careful debridement of the ulcer it can be filled with gauze packing saturated with an oily solution of azochloramide or with an 0.8 per cent solution of sulfanilamide. The dressing should be changed twice a day. Such treatment can rapidly convert a foul smelling lesion into one which is covered by clean granulations. Once this is accomplished the dressing can be changed to a vaselined gauze dressing with the anticipa-

Other methods that have been suggested for producing skin vasodilatation include the use of *typhoid vaccine* intravenously to elevate the body temperature and cause a generalized reaction. This method has the disadvantage that during the period of chill when the temperature is rising there is generalized vasoconstriction which is later replaced by vasodilatation when the temperature becomes elevated.

Another method of producing vasodilatation consists in placing the *uninvolved extremities* into hot water (40° – 45° C). This procedure is accompanied by generalized vasodilatation of arms and legs.

On first thought the application of local heat to the involved extremity would seem to be advisable since local heat causes local vasodilatation. When vessels have a limited capacity to dilate however the efficacy of such measures is dubious. Local heat accelerates all local chemical and metabolic processes increasing the circulatory demand. The measure of an adequate circulation is not the total blood flow but the total blood flow in relation to the needs of the tissue. When extremities with a limited capacity for vasodilatation are heated locally there is some vasodilatation but the elevation of temperature increases the local metabolism to a greater degree than is warranted by the amount of vasodilatation that it produces. The net effect is that of rendering the circulation less adequate. Proof of this is amply demonstrated by the ease of production of burns in an extremity with limited circulation the increase of pain in such extremities when they are heated and by the experimental evidence that gangrene of an extremity after ligation of its vessels is more frequent and more extensive if the extremity is heated and is delayed by the local application of cold.

Room temperature or at most a temperature slightly higher than room temperature but less than body temperature is perhaps ideal. The latter can be procured by using a cradle with radiant heat lights which are thermoregulated to keep any desired temperature.

If the ulcer is prevented from healing by a venous defect the return vascular flow must be accelerated. With severe secondary infection *elevation of the extremity and bed rest* are mandatory. A chronic indurated ulcer however is best treated by some ambulatory procedure such as the application of gentle but firm pressure to the entire extremity to prevent pooling of blood in dilated peripheral varicose veins. One excellent method is the use of a rubber sponge *venous heart*. The ulcer and surrounding skin are cleansed with benzene. A bland ointment is applied to the ulcer. This is covered with several layers of fluffed gauze dressing and over this four layers of sheet wadding. Finally a firm rubber bath sponge about 1 inch larger than the ulcer is applied over the dressing and held in place with a plain gauze bandage. A four inch elastic bandage is firmly applied from toe to knee with a double figure of eight about the ankle. The patient is instructed to walk. The bandage is changed every 2 to 3 days.

Another excellent ambulatory dressing is the *Unna's paste boot*. Unna's paste consists of a mixture of zinc oxide, gelatin, water and glycerin.

| | |
|------------|--------|
| Zinc oxide | 10 gm |
| Gelatin | 40 gm |
| Glycerin | 120 cc |
| Water | 140 cc |

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tion of further rapid healing. If the defect is large it may advantageously be covered with small deep grafts. The granulating surface is prepared for several days with Dakin's solution followed by a hypertonic saline dressing to make the granulations firm.

GANGRENE

When the local circulation is insufficient tissue becomes infarcted and then necrotic. This necrotic tissue is gangrenous.

Gangrene may be caused by local or systemic disturbances similar to those which produce ulcerations (p. 3986). These include *injuries* (such as burns), *pressure* (particularly in an anesthetized area), *diabetes*, *acute vascular occlusion (embolism)*, *slow vascular closure (arteriosclerosis thrombo angustis obliterans)*, *vascular spasm (ergotism)*, *trauma* to local vessels.

If the vascular disturbance is gradual the gangrene begins as a tiny black speck that may slowly progress. Sudden vascular obstruction such as that produced by an embolus may involve a large portion of the limb.



Fig. 11-1—Gangrene of foot, from circulatory damage.

The affected area becomes cold, painful and blue. Later it will become black and insensitive. At the proximal limits there slowly develops within a few days a sharp line of demarcation proximal to which there is a narrow zone of hyperemia and then normal skin.

If the lesion is uncontaminated by bacteria it slowly becomes dry and mummified and finally separates spontaneously. With secondary infection the tissues become moist and may develop a foul odor indicating the presence of gas-forming organisms. Proximal to the infected area of gangrene there is a wide zone of hyperemia, edema and cellulitis.

Treatment—*Dry gangrene* is associated with but little if any constitutional reaction. *Moist infected gangrene* is complicated by considerable fever, prostration and tachycardia. Extensive gangrene, especially if infected, is a major surgical problem. It frequently requires urgent amputation.

Small areas of dry gangrene can be treated conservatively and the limb may often be saved. In the diabetic treatment of the metabolic disturbance is essential to the successful management of the local condition.

The prognosis of gangrene is much worse in the diabetic than in the non diabetic and amputation is more often necessary in the diabetic. An area of infected gangrene in a diabetic usually requires emergency amputation. The presence of gas forming organisms likewise makes amputation mandatory.

In nondiabetics the patient may be observed for 24 to 48 hours and treated conservatively using specific chemotherapeutic agents as well as other methods to increase peripheral circulation. If however in spite of such treatment the infection spreads amputation will become necessary.

Treatment otherwise consists of measures directed toward keeping the lesion clean and dry. The lesion should always be protected with a sterile dressing which may be dry or may consist of 70 per cent alcohol and which should be changed daily under strictest aseptic precautions. At the time of the dressing the gangrenous area and surrounding skin are washed carefully with neutral soap and then dried with alcohol. Finally when demarcation is complete and the gangrenous area separates a clean ulcer will be left. The subsequent treatment is the same as for other peripheral ulcerations in the presence of peripheral occlusive arterial disease.

CHAPTER 184

MAJOR SURGERY

| | |
|--|--|
| Specialization | Preoperative Preparation |
| The Practitioner's Role in Major Surgery | Anesthesia |
| The Scope of Major Surgery | Prevention of Postoperative Discomfort and Complications |
| Surgical Indications | Active Treatment of Postoperative Discomfort and Complications |
| Surgical Risk | |
| Preoperative Investigations | |

MAJOR surgery is a therapeutic specialty which deals with the operative management of deformities injuries new growths and inflammatory processes Its scope has been extended to the correction of functional disturbances such as hyperthyroidism and hypertension and measures aimed at the relief of pain (sympathectomy) Major surgery differs from minor surgery in that its technics require more elaborate apparatus and greater training The patient is usually hospitalized the magnitude of many procedures results in an appreciable risk to life the help of trained operating room nurses surgical assistants and a special anesthetist is usually required and the technical requirements of many operative procedures warrant the services of a specially trained and qualified consulting surgeon

SPECIALIZATION

The rapid growth of surgery has necessitated the development of a variety of sub specialties and sub specialists Most large communities include an abdominal surgeon a thoracic surgeon an orthopedist a neurosurgeon an oial surgeon an otorhinolaryngologist an ophthalmic surgeon a plastic surgeon a urologist a gynecologist and an obstetrician In fact the progress of sub specialization is such that some men limit themselves to the performance of a single operative procedure such as thyroidectomy sympathectomy or herniorrhaphy

THE PRACTITIONER'S ROLE IN MAJOR SURGERY

The practitioner cooperates to the limit of his ability with his various surgical consultants He participates in diagnosis he enters into discussions of indications and optimum types of procedure he acquaints himself with gross technical details he evaluates prognosis in terms of immediate results late complications and sequels he assists with preoperative and postoperative care and he plays a dominant role in the management of medical situations that arise during the course of the surgical experience

THE SCOPE OF MAJOR SURGERY

The surgeon employs an ingenious variety of devices in order to accomplish his various purposes He may perform incision and drainage (*otomy*) he may establish a fistulous opening externally or between internal organs (*ostomy*) he may be compelled to remove an organ in whole (*ectomy*) or in part (*subtotal ectomy*) he may perform a reparative procedure

(*orrhaphy*) replace an organ (*opexy*) or execute a plastic operation (*oplasty*) for functional or cosmetic reasons

INCISION AND DRAINAGE (*otom*)

The simple procedures of incision are usually performed for evacuation of an abscess liberation of retained secretion decompression or the de-

TABLE 243.—PROCEDURES OF INCISION AND DRAINAGE (*-otomy*)

| | |
|--|--|
| Astrotomy For drainage in sinusitis (p 2125) | Mastotomy For breast abscess (p 2612) |
| Arthrotomy In suppurative arthritis (p 2905) | Myringotomy For drainage in otitis media (p 2037) |
| Cholecystotomy For hydrops of gallbladder (p 2006) | Nephrolithotomy For delivery of calculus (p 2314) |
| Choledochotomy For common duct drainage (p 2004) | Osteotomy In osteomyelitis (p 2330) |
| Cranotomy For decompression and exploration of cranial cavity (p 1429) | Pneumonotomy For lung abscess (p 2115) |
| Cystotomy For stab drainage in acute retention of urine (p 264) | Litholothotomy For delivery of calculus (p 2314) |
| Dacryocystotomy For drainage of abscess of tear gland (p 1614) | Thoracotomy For empyema (p 2219) |
| Epiotomy Preliminary to delivery | Tonillotomy For drainage in peritonsillar abscess (p 2155) |
| Glossotomy For tongue abscess | Tracheotomy To establish airway in obstruction (p 3957) |
| Hysterotomy For cesarian section (p 2694) | Tenotomy In muscle imbalance (p 1530) |
| Laryngotomy For removal of tumors of cords (p 2070) | Ureterolithotomy For delivery of calculus (p 2314) |
| | Urethrolithotomy For delivery of calculus (p 2314) |

TABLE 244.—PROCEDURES FOR ESTABLISHMENT OF THE FISTULA (*-ostomy*)

| | |
|---|--|
| Appendicostomy For drainage in enterocolitis | Entero-enterostomy In resection of small bowel (<i>side-to-side or end-to-end</i>) |
| Cholecystostomy Preparatory to removal of gallbladder (p 2006) | Enterostomy (Ileostomy) In intestinal obstruction (p 1835) |
| Cholecyst gastrostomy or -enterostomy For gallbladder drainage (p 2006) | Gastro-enterostomy In peptic ulcer (p 1763) and pyloric obstruction (p 1795) |
| Colostomy For drainage | Caecostomy In esophageal obstruction (p 1733) |
| Cystostomy Preliminary to prostatectomy (p 2448) | Jejunostomy In pyloric or gastric obstruction (p 1733) |
| Double-barreled colostomy After resection (p 1835) | |

livery of a stone (*lithotomy*) In the instance of the cesarean section *hysterotomy* accomplishes the delivery of the baby

ESTABLISHMENT OF THE FISTULA (*-ostomy*)

The surgeon establishes a fistulous opening for feeding purposes in the case of the *gastrostomy* or the *jejunostomy* *Colostomy* is intended for drainage of the fecal column when it seems desirable to protect an inflamed large bowel or when it has become necessary to close the anal orifice. Fistulous openings between loops of bowel are required to restore con-

tinuity when segments are removed because of malignant disease under these circumstances the surgeon performs *gastro enterostomy entero enterostomy* or *double barreled colostomy*

TABLE 245—I PROCEDURES FOR REMOVAL OF ORGAN (*ectomy*)

| | |
|--|--|
| Adenoidectomy | Mastoidectomy |
| For relief of infection | For infection (p 2146) |
| Appendectomy | Nephrectomy |
| For appendicitis (p 1881) | For infection or neoplasm (p 2326) |
| Cholecystectomy | Neurectomy |
| For gallbladder disease and cholelithiasis (p 1991) | For painful stumps |
| Colectomy | Oophorectomy |
| With colostomy or double barrel resection (p 1835) | For infection and neoplasm (p 2572) |
| Dacryocystectomy | Orchiectomy |
| In infection (p 1537) | For neoplasm (p 2441) |
| Embolectomy | Pancreatectomy Partial |
| For recent thrombosis (p 1123) | For hypoglycemia and malignancy (p 1943) |
| Enucleation of Eye | Parathyroidectomy Partial |
| To prevent sympathetic ophthalmia or for neoplasm (p 1559) | For relief of hyperparathyroidism (p 1233) |
| Esophagectomy | Phrennectomy |
| For malignancy of esophagus (p 1723) with jejunostomy or gastrostomy | In collapse therapy of tuberculosis (p 2199) |
| Exenteration of Orbit | Pneumonectomy |
| For malignancy (p 1559) | For abscess or malignancy of lung (p 2078) |
| Gastrectomy Subtotal | Polypectomy |
| For malignancy (p 1814) or ulcer (p 1763) with gastro-enterostomy | For relief of nasal occlusion |
| Glossectomy Partial | Prostatectomy Transurethral Perineal or Abdominal |
| For malignancy of tongue (p 1718) | For hypertrophy or neoplasm (p 2448) |
| Hemorrhoidectomy | Salpingo-oophorectomy |
| For hemorrhoids (p 3947) | For infection and neoplasm (p 2575) |
| Hysterectomy Subtotal or Radical | Sphenoidectomy |
| For neoplasm (p 2558) | For relief of infection (p 2126) |
| Iridectomy | Splanchnicectomy |
| In glaucoma (p 1578) | For relief of hypertension (p 911) |
| Laminectomy | Splenectomy |
| For exploration of spinal cord (p 1430) | For blood dyscrasias (p 1033) |
| Laryngectomy | Sympathectomy Periaxial |
| For neoplasm (p 2072) | For relief of pain (p 1394) |
| Lens Extraction | Synovectomy |
| For cataract (p 1592) | Particularly in knee joint (p 2813) |
| Lobectomy | Thymectomy |
| For abscess or malignancy of lung (p 2078) | For myasthenia gravis (p 2886) |
| Mastectomy Simple | Thyroidectomy Subtotal |
| For benign breast tumor (p 2579) | For relief of hyperthyroidism (p 1197) |
| Mastectomy Radical | Tonsillectomy |
| For malignant breast tumor includes pectoral muscles and contents of axilla (p 2581) | For relief of infection (p 2035) |
| | Turbinectomy |
| | For relief of nasal occlusion (p 2039) |
| | Varicocelectomy |
| | For varicocele (p 3933) |
| | Vesiculectomy Seminal |
| | For infection (p 2112) |

REMOVAL OF AN ORGAN (*ectomy*)

The removal of an organ is permissible when a non vital structure becomes irreparably infected (*appendectomy cholecystectomy*) for neoplastic disease particularly malignancy (*radical mastectomy subtotal gastrectomy and radical hysterectomy*) and for the correction of functional abnormalities as *splenectomy* for certain of the blood dyscrasias

subtotal thyroidectomy for hyperthyroidism and *partial pancreatectomy* in the management of hypoglycemia. Removal of the laminae of the vertebral column permits exploration of the spinal cord and sympathetic nerves and ganglia are excised for the relief of intractable pain and the control of hypertension.

TABLE 246—PROCEDURES OF REPAIR (-orrhaphy), REPLACEMENT (-opsy) AND PLASTIC SURGERY (-oplasty)

| | |
|---|--|
| Aneurysmorrhaphy | Neuroorrhaphy |
| For repair of large sac (p. 989) | For restoration of continuity |
| Angiorrhaphy | Perineorrhaphy |
| For restoration of continuity | For repair and relief of cystic and rectal |
| Arthroplasty | cele (p. 2522) |
| In chronic arthropathies (p. 2409) | Pyloroplasty |
| Colorrhaphy | For hypertrophic pyloric stenosis (p. 233) |
| In perforation (p. 133) | Repair of hard palate or cleft palate |
| Enterorrhaphy | For congenital deformity (p. 1089) |
| In perforation (p. 183) | Rhinoplasty |
| Gastrorrhaphy | Nasal plastic surgery usually cosmetic (p. 2048) |
| Suture of perforation (p. 1728) | Thoracoplasty |
| Herniorrhaphy | In tuberculosis (p. 199) |
| For epigastric, incisional, femoral, inguinal | Tenoplasty |
| hernia (p. 3091) | In muscle imbalance (p. 2509) |
| Myocardiorrhaphy | Tracheorrhaphy |
| For repair of injury | For repair of cervix (p. 252) |

TABLE 247—MISCELLANEOUS OPERATIVE PROCEDURES

| | |
|--|---|
| Amputation of Extremities | Ligation of Jugular Vein |
| (See p. 3254) | In sinus thrombosis (p. 2149) |
| Arthrodesis | Ligation of Saphenous Vein |
| In arthropathies (p. 2409) | For thrombophlebitis and varicose veins (p. 1123) |
| Corneal Transplantation | Isthmectomy and Lithotomy |
| In keratitis (p. 1626) | For crushing and removal of calculus (p. 2314) |
| Correction of Muscle Imbalance | Pericardiectomy |
| In strabismus (p. 1530) | In constrictive pericarditis (p. 1010) |
| DENERVATION OF CAROTID SINUS | Reduction of Dislocations and Fractures |
| In carotid sinus syncope (p. 922) | By open and closed method (p. 2990) |
| Effluoration | Submucous Resection |
| For bladder neoplasm (p. 922) and hydro- | In nasal occlusion (p. 2048) |
| cephalus (p. 1409) | Transplantation of Ureters |
| Fenestration | In malignancy of bladder (p. 233) |
| For otosclerosis (p. 2095) | Vascularization of Pericardium |
| Ligation of Ductus Arteriosus | In coronary occlusion (p. 983) |
| In congenital anomaly (p. 937) | |

REPAIR (-orrhaphy), REPLACEMENT (-opsy) AND PLASTIC SURGERY (-oplasty)

Operative procedures that call for repair, replacement or plastic changes may be performed for mere cosmetic reasons (*rhinoplasty*) but they may have much greater significance as illustrated by *pyloroplasty* in hypertrophic pyloric stenosis and *thoracoplasty* to control pulmonary tuberculosis.

MISCELLANEOUS OPERATIVE PROCEDURES

In Table 247 are listed a number of ingenious operative procedures. Some are relatively simple as the *submucous nasal resection*; others are

tinuity when segments are removed because of malignant disease under these circumstances the surgeon performs *gastro enterostomy entero enterostomy* or *double barreled colostomy*

TABLE 24.—PROCEDURES FOR REMOVAL OF ORGAN (*ectomy*)

| | |
|--|--|
| Adenoidectomy | Mastoidectomy |
| For relief of infection | For infection (p 2146) |
| Appendectomy | Nephrectomy |
| For appendicitis (p 1881) | For infection or neoplasm (p 2326) |
| Cholecystectomy | Neurectomy |
| For gallbladder disease and cholelithiasis (p 1991) | For painful stumps |
| Colectomy | Oophorectomy |
| With colostomy or double barrel resection (p 1835) | For infection and neoplasm (p 2073) |
| Dacryocystectomy | Orchiectomy |
| In infection (p 1557) | For neoplasm (p 2141) |
| Embolectomy | Pancreatectomy Partial |
| For recent thrombosis (p 1123) | For hypoglycemia and malignancy (p 1913) |
| Eucleation of Eye | Parathyroidectomy Partial |
| To prevent sympathetic ophthalmia or for neoplasm (p 1559) | For relief of hyperparathyroidism (p 1233) |
| Esophagectomy | Phrenicectomy |
| For malignancy of esophagus (p 1723) with jejunostomy or gastrostomy | In collapse therapy of tuberculosis (p 2139) |
| Exenteration of Orbit | Pneumonectomy |
| For malignancy (p 1559) | For abscess or malignancy of lung (p 2078) |
| Gastrectomy Subtotal | Polypectomy |
| For malignancy (p 1814) or ulcer (p 1763) with gastro-enterostomy | For relief of nasal occlusion |
| Glossectomy Partial | Prostatectomy Transurethral, Perineal or Abdominal |
| For malignancy of tongue (p 1718) | For hypertrophy or neoplasm (p 2448) |
| Hemorrhoidectomy | Salpingo-oophorectomy |
| For hemorrhoids (p 3947) | For infection and neoplasm (p 2575) |
| Hysterectomy Subtotal or Radical | Spheno-ethmoidectomy |
| For neoplasm (p 2558) | For relief of infection (p 2126) |
| Iridectomy | Splanchnicectomy |
| In glaucoma (p 1578) | For relief of hypertension (p 211) |
| Laminectomy | Splenectomy |
| For exploration of spinal cord (p 1430) | For blood dyscrasias (p 1053) |
| Laryngectomy | Sympathectomy Periarterial |
| For neoplasm (p 2072) | For relief of pain (p 1394) |
| Lens Extraction | Synovectomy |
| For cataract (p 1592) | Particularly in knee joint (p 2813) |
| Lobectomy | Thymectomy |
| For abscess or malignancy of lung (p 2078) | For myasthenia gravis (p 2886) |
| Mastectomy Simple | Thyroidectomy Subtotal |
| For benign breast tumor (p 2579) | For relief of hyperthyroidism (p 1197) |
| Mastectomy Radical | Tonsillectomy |
| For malignant breast tumor includes pectoral muscles and contents of axilla (p 2581) | For relief of infection (p 2038) |
| | Turbinectomy |
| | For relief of nasal occlusion (p 2039) |
| | Varicocelelectomy |
| | For varicocele (p 3933) |
| | Vesiculectomy Seminal |
| | For infection (p 2115) |

REMOVAL OF AN ORGAN (*ion y*)

The removal of an organ is permissible when a non vital structure becomes irreparably infected (*appendectomy cholecystectomy*), for neoplastic disease particularly malignancy (*radical mastectomy subtotal gastrectomy and radical hysterectomy*) and for the correction of functional abnormalities as *splenectomy* for certain of the blood dyscrasias

tomy and radiation therapy may prove much safer and quite as satisfactory as radical mastectomy with its high operative mortality

Multiple Stage Operations—In certain instances graded or multiple stage procedures are safer than a one stage operation. The patient suffering from acute cholecystitis with cholelithiasis may survive cholecystostomy but not a cholecystectomy. Under such circumstances the gallbladder may be removed at a later date when conditions are more favorable. The multiple stage subtotal thyroidectomy is far safer in the treatment of hyperthyroidism (p 1197)

Better a live lamb than a dead lion

SURGICAL RISK

Surgical risk is estimated by adding the sum of the *operative hazards* as estimated by the surgeon to the *individual patient risk* as estimated by the practitioner

Operative Risk—The surgeon should not object if the general practitioner questions him bluntly concerning his personal experience and fortunes with the particular procedure that is under discussion. These questions are of course waived in immediate operations of necessity. They are considered carefully in deferred operations whether of necessity or choice.

The practitioner is entitled to know of his surgeon how many similar procedures he has done, approximately how many of the operative patients have succumbed to the technical procedure, the frequency, degree and duration of complications, invalidism and incapacitation.

References to the literature are interesting but not pertinent. The individual patient faces the risk with his surgeon, not with a collective abstract.

Factors Entering into the Individual or Patient Risk—The individual patient risk is estimated by the general practitioner. It depends upon a wide variety of factors both physiologic and pathologic.

Age—Surgical risk is greatest at the extremes of life. Only *immediate emergency surgery* is permissible in infants and elderly individuals. To be sure these often stand procedures amazingly well but wherever possible conservatism is the rule.

Newborn infants with congenital abnormalities that threaten life (such as an imperforate anus) and somewhat older children with hypertrophic pyloric stenosis (p 2735) often must be subjected to surgery as a *life saving measure*. When the need is not so imperative as in cleft palate operation is deferred until nutrition is better maintained and equilibrium has been established.

Emergency surgery unfortunately must often be performed on the *aged*. Only the simplest procedure should be executed. Thus for the old gentleman with retention of urine from prostatic obstruction initial cystostomy would be preferable to a radical prostatectomy.

In the elderly malignancies are usually slow growing. It is better to have a live patient with a jejunostomy or colostomy than a dead one on whom radical excision has been successfully performed.

I likewise in the elderly the choice of anesthesia presents a difficult problem. *Spinal anesthesia* is hazardous since the rapid alteration in blood pressure may lead to vascular accidents. Nevertheless in many institu-

required as life saving devices (*pericardiolysis* ligation of the ductus arteriosus and *amputations* of the extremities)

SURGICAL INDICATIONS

Immediate Operations of Necessity—The immediate operation of necessity is of course performed to save life. There is no time for discussions of risk or other details in dealing with the patient who has an ectopic pregnancy, a gangrenous intestinal obstruction, perforation of a hollow viscus, or a tension pneumothorax. Once the diagnosis is established the surgical procedure is performed as rapidly as possible with what implements are at hand.

Patients who refuse operations of urgent necessity do so at their own risk. The surgeon and practitioner must be absolved from any responsibility.

The only permissible delay is one of an hour or two to permit *restoration of blood volume* in severely dehydrated patients or to *combat moderate or severe shock* (p. 937).

Deferred or Delayed Operations of Necessity—Operations of necessity may be deferred for a few days or weeks in order to permit the patient to put his affairs in order, to improve the general nutrition, and to prepare the operative site.

Operations of Election—Operations of election or choice include the *repair of hernia*, the *removal of a benign tumor* such as a uterine fibroma or an ovarian cyst, *cosmetic surgery*, the *correction of disabilities and deformities*.

The operations of election or choice must be carefully adjudicated before any decision is made. *Operative risk* (p. 3995) is weighed against possible *operative gain*, and the situation is carefully explained to the patient so that he is in full possession of the details before making his decision.

In general operations of election or choice should not be done if the *risk approaches 1 per cent*. An exception to this rule is the condition of possible malignancy. The practitioner more readily agrees to operation for gastric ulcer where malignancy is prone to occur than to that for duodenal ulcer where malignancy is virtually unknown.

The patient who travels should have a hernia repaired and a grumbling appendix removed. Patients who live at long distances should not hazard an acute surgical exigency. Repair of a hernia would be more reasonable in a young active individual than in an elderly sedentary patient.

It is axiomatic that if the *hazard involved in surgical therapy is greater than that of the disease itself*, operation is illogical. At times considerable judgment must be exercised in deciding whether or not operation should be performed.

Type of Procedure—If an operation of choice has been decided upon the type of procedure is discussed. For example, in order to remove a large carcinoma of the rectum, an extensive abdominoperineal resection of the entire rectum and sigmoid colon is necessary. If the patient is elderly and feeble, it may prove wiser to employ a conservative palliative procedure like *colostomy* rather than face an operative risk that may exceed 50 per cent. In carcinoma of the breast in an elderly individual, *simple mastec-*

individuals who have a malignancy it is better to postpone operation until anhydremia, hypoproteinemia, anemia and vitaminosis have been relieved

Fever—Certain of the infectious diseases (pneumonia typhoid fever and ulcerative colitis) are often complicated by conditions that require urgent surgery. The risk is naturally greater in these febrile patients who are depleted by their underlying disease

In an emergency local anesthesia is employed only the minimal necessary technical maneuvers being carried out. The patient is sustained preoperatively and postoperatively by infusion and transfusion

Diabetes—Surgery in the diabetic is hazardous the difficulties being both surgical and metabolic (p 1259)

To avoid both difficulties the surgeon is wise if a *continuous intravenous infusion* (p 3375) is established before any type of surgical procedure is attempted. The infusion should be made with 5 per cent dextrose in physiological saline solution. At least 1 unit of insulin is added to the drip for each 2 gm of carbohydrate. Urine samples are examined at each voiding for sugar and ketone bodies. In the presence of glycosuria additional insulin is given. For acidosis the concentration of sugar is increased to 10 per cent with insulin in the ratio of 1 unit per gram. The vehicle is changed to a buffered solution such as sodium lactate or Hartman's solution

Tuberculosis—The tuberculous are of course subject to the development of the usual surgical conditions and in addition may face operative treatment of the specific lesion (thoracotomy nephrectomy etc)

Patients with tuberculosis and other chronic pulmonary diseases must be especially guarded with reference to *anesthesia*. If it is at all possible *local anesthesia* should be employed (p 272)

Cardiac Invalidism—The youthful cardiac invalid is commonly incapacitated as the result of a rheumatic valvulitis or congenital cardiac abnormality. When it is necessary to operate on the young the risk should be gauged not by murmurs and the extent of the valvulitis but by the physiological evidences of circulatory efficiency (p 199). Operations of choice should be deferred. Operations of necessity may be carried out confidently if there are no evidences of circulatory failure

It is to be remembered that *tonsillectomy* in children with rheumatic valvulitis as well as *removal of teeth* may precipitate *subacute bacterial endocarditis* (p 286). Post tonsillectomy polyomyelitis (p 465) has also occurred. Hence these procedures should be done with the greatest concern. It is wise to employ penicillin as an ante and post operative systemic prophylactic

Rheumatic children who have previously been on doses of *digitalis* should be maintained with that drug. Digitalis of course should not be given if the rhythm of the heart is regular and there are no evidences of decompensation. Under these circumstances it is more likely that the drug will be toxic than helpful (p 858)

In *older patients* emergency surgery must often be done in the face of hypertension arteriosclerosis coronary narrowing or closure. The elderly prostatic often suffers from all three of these difficulties. Naturally only emergency surgery is to be done when there is advanced vascular disease. Local infiltration anesthesia or ethylene should be employed

tions prostaticectomy upon the aged patient is routinely performed under low spinal anesthesia with remarkably low mortality. The irritating gaseous anesthetics particularly ether are prone to set up pneumonitis. Local anesthesia with preoperative barbiturate sedation is the method of choice. With a specialist anesthetist ethylene or cyclopropane is highly desirable.

Following operation in the aged *prolonged rest in bed* invites hypostatic pneumonia and phlebitis. *Semle and exhaustion psychoses* often complicate a surgical experience (p 1382).

Despite all these ominous warnings old people often go through a surgical procedure of considerable magnitude without untoward complications or sequels. These happy experiences must not delude the practitioner or the surgeon into excessive optimism in other instances.

Pregnancy—While elective surgery should be deferred during pregnancy nevertheless a gravid woman commonly tolerates emergency surgery such as appendicectomy with remarkable fortitude and the experience is often happily uneventful.

Acute appendicitis (p 1881) incidentally is often difficult to diagnose during pregnancy and removal of the inflamed viscus may be technically difficult. Despite the necessary manipulation of the uterus the fetus is rarely disturbed and pregnancy is not interrupted.

Obesity—Obesity adds significantly to the technical difficulties and risks of operation. In abdominal surgery the thickness of the panniculus and fat deposits in the omentum are a real menace. *Hernia repair* is difficult and often unsuccessful in stout patients. Patients who are overweight stand the anesthetic poorly. They are prone to develop *postoperative complications* particularly of the respiratory and digestive systems.

Whenever possible operation on the obese patient should be deferred until there has been a significant reduction in weight. This is particularly true in the surgery of diseases of the gallbladder which are of common occurrence in the obese (p 1989).

Alcoholism—Alcoholism adds to all the difficulties of surgery the patient takes the anesthetic poorly and reacts badly and atypically to drugs. Topers have not the temperament nor the resistance to be good patients. Rapidly deprived of alcohol they tend to develop *delirium tremens*. Consequently doses of alcohol should be given throughout the first few postoperative days. Thiamin chloride and ascorbic acid are administered intravenously and nicotinamide intramuscularly because of the frequent occurrence of an avitaminosis (p 616).

In dealing with alcoholics it is important to remember that *alcoholic gastritis* may give all of the manifestations of an intra abdominal accident such as the penetration or perforation of an ulcer. Laparotomy reveals no abnormality to the chagrin of all concerned. The risk of operation has been an unnecessary hazard.

Contrariwise alcoholism may obscure a surgical catastrophe (p 1748). The patient succumbs to peritonitis following appendicitis or to visceral rupture under the mistaken diagnosis of alcoholic gastritis. In skull fracture with intracranial complication the coma is obscured by the inebriation.

Malnutrition and Cachexia—Every effort should be made to defer operation on patients whose nutrition has been undermined. Even with

TABLE 219.—PREOPERATIVE INVESTIGATION

| |
|---|
| Routine History (p 3470) |
| Mandatory before any type of procedure |
| Routine Physical Examination (p 3476) |
| Mandatory before any type of procedure |
| Complete Hemogram (p 3684) |
| Mandatory before any type of procedure |
| Serologic Tests for Syphilis |
| Mandatory before any type of procedure |
| Urinalysis (p 3666) |
| Mandatory before any type of procedure |
| Renal Function Tests (p 3686) |
| In presence of albuminuria, cylindruria or pyuria or history of nephropathy, neuropathy or hypertension |
| Ketone Bodies in Urine (p 3680) |
| With glycosuria or history of diabetes. After protracted fluid loss by vomiting, diarrhea or hemorrhage |
| Chest X-ray (p 3440) |
| For lung fields in presence of respiratory symptoms or signs or with history of tuberculosis. For size and configuration of heart after middle age and in patients with circulatory symptoms or signs or hypertension |
| Electrocardiogram (p 805) |
| Routine after midline and in patients with circulatory symptoms or signs or hypertension |
| Blood Grouping (p 3408) |
| Routine in presence of anemia, hemorrhagic diathesis (p 1109) and those who face serious, prolonged or shocking operative procedures |
| Blood Sedimentation Rate (p 3407) |
| Before operations in infectious states |
| Blood Coagulation Studies (p 3406) |
| Routine in jaundice (p 191) and preceding surgery of liver, biliary passages, pancreas and spleen. Advisable prior to any operation before which patient has bled |
| Pregnancy Test (p 2438) |
| Routine before any pelvic operation in patient who has uterine enlargement or pelvic tumor |
| Bladder Catheterization (p 4936) |
| Routine before operation for pelvic tumor |
| Iso-osmotic Urography (p 2431) |
| Routine before operations on ureters, renal pelvis or kidney |
| Endotracheal Intubation (p 3004) |
| Preceding any operation on neck, but particularly subtotal thyroidectomy |

carried out in the period between the decision to operate and transfer to the operating room. In deferred operations there is sufficient time to im-

Few clinical experiences are more extraordinary than the ease with which the older cardiac invalids go through a surgical procedure. However their course may be complicated by cerebral apoplexy, coronary thrombosis, peripheral phlebitis, thrombosis and embolism, hypostatic pneumonia or an exhaustion psychosis.

See *Differential Diagnosis of Febrile Disorders in the Aged* (p 980).

Nephritis—Contrary to general belief, operative procedures including anesthesia are not particularly trying on renal function. Except with marked kidney insufficiency, difficulty should not be encountered.

Surgery of the urinary organs themselves may be complicated by reflex anuria (p 2232). For this reason, a continuous intravenous drip is initiated prior to operation.

Psychoses—Surgery is attended with *great risk* in the psychotic, post-operative psychoses often being fatal. These unfortunates are the patients who leap from windows, tear dressings and drains from wounds and otherwise make a nightmare of a surgical procedure. They require continuous nursing care.

The Types of Individual Patient Risks in Surgery—After analysis of the factors that enter into the estimation of the surgical hazard, patients may be classified as good, fair or poor risks.

GOOD RISKS—The good surgical risk is the healthy patient between the ages of 12 and perhaps 45, whose entire physical examination is normal, whose habits are exemplary and whose emotional and psychic functions are well balanced. Such patients conscientiously may be advised to undergo procedures of election or choice.

FAIR RISKS—The patient with a fair surgical risk is one who has organic damage but a reasonably wide reserve of physiologic function. Thus a child or an adult with mitral stenosis and good compensation, a patient with healed tuberculosis of an apex, a middle-aged or elderly patient with a moderate hypertension and mild glycosuria might well be called fair risks.

Surgery of election should be avoided wherever possible in the fair risk patient. It may be deferred until the underlying difficulty is better controlled. The operation may be staged, as for example in subtotal thyroidectomy or the removal of a colonic neoplasm.

POOR RISKS—The poor risk patient is one whose reserve has been broken down or whose reserve threatens to break down under the added strain of operative and anesthetic trauma.

These patients should never be subjected to an operation of choice. If it is necessary to do urgent surgery, minimal manipulation is permissible.

PREOPERATIVE INVESTIGATION

Before permitting any major surgical procedure, the general practitioner should perform a *routine examination* and such additional *special tests* as may be indicated by the condition of the patient or the nature of the surgical procedure.

PREOPERATIVE PREPARATION

The extent of preoperative preparation is mostly determined by the nature of the procedure. In an acute emergency, only the essentials are

been described in the section on Minor Surgery (p 3913) Under institutional conditions the practitioner has also the choice of employing the more complicated techniques required for the administration of nitrous oxide ethylene and cyclopropane These may be supplemented with curare (p 3888)

Nitrous Oxide (N_2O)—Nitrous oxide (‘laughing gas’) is the most frequently employed of the gaseous anesthetics In expert hands it has a wide margin of safety It is pleasant to take and produces rapid loss of consciousness

Advantages—The great advantage of nitrous oxide is the rapid and complete recovery Within a few moments the patient is restored to normal and may if the surgical condition permits get out of bed and even leave the hospital or the office It is less irritating to the liver than chloroform or ether It is used preferentially in patients suffering from chronic degenerative hepatic disease

Disadvantages—Nitrous oxide has distinct limitations It is unsatisfactory for prolonged operations and in intra abdominal procedures in which there is considerable manipulation of viscera Ether usually must be added at least for a time to secure better relaxation

Because of its depressant action on heart muscle and the compulsory anoxemia attendant upon its use nitrous oxide is avoided in patients who suffer from circulatory difficulties

Ethylene—Ethylene has all of the advantages of nitrous oxide and possesses its disadvantages to a lesser degree *Induction* is somewhat more prolonged but easy and pleasant *Recovery* is slightly less rapid but equally agreeable The *maintenance of anesthesia* is more easily accomplished particularly by an expert The effect may be prolonged for protracted procedures Relaxation is satisfactory

Unfortunately ethylene is *highly explosive* and several patient deaths have occurred due to sparks from static electricity Because of this danger ethylene should not be used except by the expert anesthetist under carefully controlled institutional conditions where provision has been made to prevent electrical accidents

Cyclopropane—Cyclopropane is an extraordinary innovation in the armamentarium of the anesthetist It is a hydrocarbon gas with the empirical formula C_3H_6 It produces complete and rapid relaxation even when administered in small quantities As little as 10 to 20 per cent concentration of the drug induces surgical anesthesia The remaining gas administered consists of oxygen The inclusion of this large quantity of oxygen makes cyclopropane the ideal anesthetic for patients suffering from respiratory disease or any type of abnormality resulting in a lowered vital capacity

The *disadvantages* of cyclopropane are threefold (1) it is *highly explosive* (2) it is a *cardiac irritant and depressant* (3) because of its great potency *overdosage* may occur

Cyclopropane must be administered with the greatest care by a specialist anesthetist It should be employed only in institutions that are safeguarded against the risk of explosion The addition of helium to the anesthetic mixture of cyclopropane and oxygen reduces the explosion hazard

It may be said that in expert hands cyclopropane is the safest anes

prove the general health of the patient eliminate possible untoward factors and attempt to anticipate or prevent postoperative difficulties

TABLE 249—PREOPERATIVE PREPARATION

Get Signed Permission for Operation

From patient or next-of kin from parents or guardians of minors

Dental Hygiene (p 1639) and Care of Nose and Throat

Before delayed operations particularly those involving oropharynx and respiratory tract to avoid postoperative pneumonitis (p 2189) and abscess of lung (p 2215)

Remove Artificial Teeth and Dentures

To prevent aspiration and impaction

Shave Local Site

Do surgical prep day in advance and bandage in deferred procedures

Preoperative Chemotherapy

With penicillin (p 106) soluble sulfonamide (p 88) or streptomycin (p 104) for systemic prophylaxis with oral streptomycin or insoluble sulfonamide preparatory to bowel surgery

Leg Exercises

Before delayed operations particularly for flabby and obese to prevent postoperative phlebitis (p 1123)

Gastric Lavage

Before stomach surgery (p 1758)

Vaginal Douche

Before gynecologic procedures (p 2599)

Colonic Irrigations

Before surgery of lower bowel (p 1835)

Weight Reduction (p 695)

Before delayed operation particularly laparotomy

Weight Gain (p 671)

Before delayed operation particularly in hyperthyroidism (p 1137)

Hypnotic (p 3830)

At bedtime eve of operation

Empty Bladder

Avoid catheterization if possible use urinal in bed for practice

Empty Bowels

Use suppository or soapuds enema Catharsis favors postoperative distention (p 4010)

Basal Anesthesia (p 3913)

Double or triple hypnotic dose orally followed by morphine or demerol with scopolamine or avertin with amylene hydrate by rectum

Continuous Intravenous Drip (p 3375)

Of 5 per cent dextrose in saline for protracted or shocking procedure Substitute plasma if necessary Add citrated blood for significant blood loss Cover dextrose with insulin (1 Unit for each 1 or 2 gm) in diabetics

ANESTHESIA IN MAJOR SURGERY

Major surgery requires anesthesia except in the comatose or unconscious patient Most of the methods of local and general anesthesia have

probing of the wound a small or moderately large amount of clear yellowish fluid is expressed. This so called serum is an exudate which has accumulated as an inflammatory response to large amounts of suture

TABLE 250—PREVENTION OF POSTOPERATIVE DISCOMFORT AND COMPLICATIONS

Back Pain

Support back to prevent buckling during transfer from bed or stretcher to and from operating table

Aspiration and Asphyxia

On return from operating room elevate foot of bed while patient lies flat, without pillow. Turn head to one side and support chin in elevated position. Grasp tongue if necessary. Insert air way if required. Prepare suction apparatus to remove secretions and vomitus.

Dehydration

Do not overheat room. Do not wrap patient in heavy blankets. Ventilate room and use sheet and one thin blanket. Give intravenous drip until fluids can be taken by mouth.

Pulmonary Stasis and Congestion

Turn patient from side to side at frequent intervals. Produce hyperpnea with inhalations of 5 or 10 per cent carbon dioxide. Encourage deep breathing as soon as consciousness returns. Early ambulation (p. 412).

Stasis in Extremities and Thrombophlebitis

Insist upon contraction and voluntary movement of leg muscles at hourly intervals after return of consciousness. Early ambulation (p. 412).

Abdominal Distention

Avoid preoperative purge. Substitute demerol for morphine. Start intramuscular injections of 1 cc of 1:4000 neostigmine postoperatively and continue at 4 or 6 hour intervals until gas is freely passed. Increase dose to 1 cc of 1:2000 if weaker solution is ineffectual or there is obvious ileus. Insert rectal tip just after injection. Encourage taking of warm fluids and soft diet at earliest possible moment to reestablish peristaltic gradient. Early ambulation (p. 412).

Nausea and Vomiting

Avoid ether in favor of ethylene or cyclopropane. Substitute demerol for morphine. Avoid cold and iced drinks.

Shock

Prepare for plasma infusion if shock is anticipated. Substitute citrated blood if there has been significant blood loss. Use liberal doses of opiate or demerol to ease pain and allay restlessness and anxiety.

Infection

Topical or systemic use of penicillin.

Thrombosis

Heparinize (p. 100) if intravascular clotting is feared. Early ambulation (p. 412).

Bleedings

Turn frequently. Smooth sheets. Protect skin by rubbing, drying and application of talc.

material particularly catgut or to large amounts of necrotic tissue that have been strangulated by too tight ligation.

Hematomas in wounds are due to inadequate hemostasis or slipping of a ligature. If superficial they usually manifest the same signs as reten-

thetic for the poor risk patient In inept hands it is one of the most dangerous anesthetics for any patient

THE CHOICE OF ANESTHETIC IN SURGERY

The choice of anesthetic in major surgery rests almost exclusively with the operating surgeon and the anesthetist Some men are particularly expert in the use of *local or spinal anesthesia* They prefer to employ this modality Others have a preference for *inhalation anesthesia*

The practitioner errs if he interferes with the customs and habits of his consultant

The Practitioner Anesthetist—When the practitioner is called upon to serve as anesthetist for his own patient he should use that form of anesthesia with which he is most conversant *Ether* is the safest inhalation anesthetic The preliminary use of *gas and oxygen* to ease induction should be part of the technical equipment of every general practitioner

Unless he has special training the practitioner should not employ ethylene or cyclopropane

The Specialist Anesthetist—The *bad risk patient* is entitled to the services of an expert *specialist anesthetist* (p 3899) The practitioner should not interfere with the type of anesthesia chosen by the expert Some of the specialists have a preference for ethylene Others would rather use cyclopropane Many of the older men still use nitrous oxide and are able to maintain satisfactory surgical anesthesia for long periods of time

PREVENTION OF POSTOPERATIVE DISCOMFORT AND COMPLICATIONS

Many of the postoperative discomforts and complications may be averted by meticulous attention to the preoperative preparations else where outlined (p 4002) Efforts at prophylaxis are continued immediately upon the patient's return from the operating room by the additional measures shown in Table 250

THE ACTIVE TREATMENT OF THE COMMONER POSTOPERATIVE COMPLICATIONS

A variety of generic postoperative complications may be anticipated following the completion of any major surgical procedure Most often these disturbances are local systemic digestive respiratory urinary or circulatory Added difficulties arise in the performance of certain of the highly specialized procedures Thus an aseptic pyrexia is frequent following sub total thyroidectomy tetany may be encountered after removal of the parathyroid glands and leakage along the suture line is a constant hazard in the surgery of the hollow bowel These special complications are dealt with more properly in discussions of the local surgical procedures

LOCAL COMPLICATIONS

Slight induration of the wound or adjacent tissues unaccompanied by other symptoms or signs may merely indicate a reaction to suture material The induration may mask a deep accumulation of serum blood or pus It may subside with or without discharge of a piece of suture material through the wound A variable number of wounds become swollen after four to seven days There are no associated signs of inflammation On gentle

separation of the wound with the prolapse of loops of intestine *immediate suture* under anesthesia is the most satisfactory method of treatment

If the patient's condition is poor the wound should be packed and strapped *Secondary suture* is attempted when the patient's condition permits Before and after operation massive doses of penicillin are injected intramuscularly in aqueous solution in oil wax suspension (p 106)

In any event abdominal distention is reduced or prevented by the use of the indwelling stomach tube with continuous suction and the other methods described in the treatment of ileus (p 4010)

ACTIVE TREATMENT OF POSTOPERATIVE COMPLICATIONS

Shock Primary or Delayed (p 920)

Establish plasma infusion Sedate with opiate or demerol Prepare for blood transfusion (p 3778)

Hemorrhage

Summon surgeon for packing or hemostasis under anesthesia if necessary institute intravenous drip of 5 per cent dextrose in saline add plasma if shock impends until cross matched citrated blood can be obtained Do not fear dislodgement of clot from bulk of infusion or transfusion

In presence of jaundice give bile salts orally and inject menadiolone (vitamin K) 1 or 2 mg ($\frac{1}{64}$ or $\frac{1}{32}$ gr) intramuscularly

Dehydration

Continue or initiate intravenous drip of 5 per cent dextrose in saline or distilled water until daily urinary output reaches 1500 cc

Acidosis or Electrolyte Imbalance (p 721)

Substitute $\frac{1}{6}$ molar sodium chloride Ringer solution in intravenous drip

Hypoproteinemia (p 708)

Establish plasma infusion or citrate transfusion

Avitaminosis (p 616)

Add thiamin chloride 100 mg ($\frac{1}{2}$ gr) riboflavin 5 mg ($\frac{1}{12}$ gr) and ascorbic acid 200 mg ($\frac{1}{4}$ gr) to intravenous drip

POSTOPERATIVE FEVER

A postoperative elevation of temperature accompanies most major procedures The febrile rise may be due to *septic* or *aseptic* causation the detection of the exact origin is of paramount importance in the establishment of the therapeutic regimen The commoner definitive causes are indicated in Table 251 At times despite best efforts the cause remains obscure and the temperature curve flattens out without the practitioner being any the wiser While this state of blissful ignorance does not represent the ideal in medicine it is nevertheless a source of intense satisfaction

tion of serum. If deep in the muscular layers there may be a rather large area of induration. Hematomas should not be disturbed unless extensive. After five to seven days the hematoma secondarily liquefies and can be evacuated through a syringe or by inserting a probe through the lower angle of the wound.

If the hematoma is deep in the muscle layers the patient is kept in bed for at least five days longer than the usual postoperative period. This permits healing of the wound edges separated by the clot.

Superficial wound infection is diagnosed by the redness, swelling and tenderness present on or about the local area. Crepitation may be felt with an anaerobic contaminant (p. 300). The first sign of wound infection may be a sudden or persistent slight pyrexia.

Deeper wound infection may occur with a seemingly clean wound. Particularly in laparotomy it is possible to have a good superficial wound with extensive underlying intraperitoneal infection. Under these circumstances there are the usual signs of peritoneal irritation. Tenderness and rigidity may be elicited. Usually there will be a febrile disturbance with a leukocytosis. While the peritoneal infection is commonly just beneath the operative site the inflammatory process may extend upward along the lumbar gutter to the *subphrenic regions*. An early sign of intraperitoneal infection is an attack of unexplained diarrhea (p. 1840). With pelvic infection the inflammatory mass may be palpable by rectum or by vagina.

The course of the peritoneal infection may pursue one of three trends: (1) The process may *resolve itself* spontaneously. (2) An abscess may be formed which *spontaneously bursts* into the wound or the hollow bowel discharging itself with the feces. (3) A *localized abscess* may form in the subphrenic or pelvic regions requiring incision and drainage.

Wound infection may necessitate the *early removal of sutures*. Under these circumstances the wound is weakened. Any sudden strain results in a separation of the layers of the abdominal wall. This separation may involve the superficial skin or the deeper layers of the fascia and muscle.

In patients whose course has been stormy from the beginning particularly if the abdomen has been distended and there has been retching from nausea and vomiting the wound appears puffy and prominent. There may be a persistent discharge of thin yellowish or pinkish fluid. Palpation reveals local tenderness. The patient complains of persistent and localized pain. When one or two of the sutures are removed the entire skin edge separates. A loop of bowel or a mass of omentum is found lying between the layers of the abdominal wall. This is the so called *subcutaneous type of wound rupture*.

Wound rupture is most apt to occur in the elderly, the obese, those who are undernourished and in patients with malignant disease. Upper abdominal wounds are more apt to disrupt than lower abdominal wounds. The vertical type of incision is more apt to be disrupted than the transverse incision. Muscle splitting and midline incisions more frequently rupture than muscle retracting and gridiron incisions. The tendency to rupture is also dependent upon the type of closure which is a matter of surgical technique.

Treatment of Wound Disruption—The treatment of wound disruption is surgical. If the patient is in good physical condition and there is wide

an intestinal obstruction or a peritonitis. These complications are elsewhere considered (p 1655).

Hiccough (Singultus)—Hiccough is the result of repeated clonic spasms of the diaphragm associated with closure of the glottis. It may be due to reflex, toxic, psychogenic and central causes.

See *Differential Diagnosis of Hiccough* (p 1933).

Postoperative hiccough usually is of *reflex* origin. It is most apt to be noted after operations in the upper abdomen upon such organs as the stomach, colon and gallbladder. In individuals with impaired renal function hiccough of *toxic* origin may be one of the first evidences of nitrogen retention and impending uremia. This is particularly common following operations upon the genito-urinary tract. *Psychogenic singultus* although not frequent follows operations of any type even those of a minor character in highstrung emotional individuals. The *central* type of hiccough is noted at times following operations upon the brain and spinal cord.

An attack of hiccough may last for a few hours or for days or even weeks. Under the former circumstances it may cause little annoyance. When prolonged it may result in exhaustion and even death. Singultus coming on quickly after operation is apt to be of reflex or neurotic origin. It is usually of short duration. Hiccough coming on after several days is more likely to be of toxic origin and therefore is usually more ominous.

Numerous remedies have been advocated for the *treatment* of postoperative hiccough. In instances of reflex and neurotic origin various *physical measures* have been recommended. These include holding the breath while slowly drinking a glass of water, traction upon the tongue, pressure over the eyeballs, pressure over the side of the neck in the ap proximate region of the phrenic nerve, procaine block of the phrenic nerve.

In addition to the use of physical measures *drug therapy* includes the use of antispasmodics, sedatives and opiates. The patient is usually dosed and often overdosed with barbiturates, bromides, chloral, morphine, paraldehyde, chloroform, water, Hoffmann's anodyne, benzyl benzoate, belladonna, aminophylline and avertin.

One of the most effective and least noxious methods consists in the inhalation of *carbon dioxide*. This is performed most simply by holding the patient's nose and having him breathe back and forth into an ordinary paper bag held snugly about the mouth to prevent leakage. As the patient rebreathes the concentration of carbon dioxide within the bag increases rapidly. This produces a central effect which usually causes prompt cessation of hiccough. If symptoms recur treatment is repeated as frequently as necessary. If desired the carbon dioxide is administered through a snugly fitted face mask which is connected with a tank containing a mixture of 5 per cent carbon dioxide and 95 per cent oxygen.

In cases of persistent hiccough when the various procedures outlined above fail a *nasal catheter* is passed into the stomach in order to determine the presence of *gastric retention*. This is of considerable importance because hiccough may be the precursor of vomiting in adynamic ileus, gastric dilatation, intestinal obstruction and peritonitis. If gastric retention is present the nasal tube is left indwelling and fluids are administered intravenously. Such treatment usually results in prompt cessation of symptoms.

COMPLICATIONS INVOLVING THE DIGESTIVE SYSTEM

The postoperative complications involving the digestive system include nausea and vomiting hiccough adynamic ileus gastric dilatation intestinal obstruction and parotitis

Nausea and Vomiting—Nausea and vomiting occur frequently for the first three or four post anesthetic hours. No treatment is required other than the maintenance of quiet the administration of a sedative or demerol and occasional sips of fluid to moisten the tongue and mouth.

With persistent nausea it may be wise to induce vomiting by the administration of a glass of warm water containing a heaping teaspoonful of bicarbonate of soda. The emesis acts as a gastric lavage and tends to

TABLE 251—ETIOLOGY AND TREATMENT OF POSTOPERATIVE FEVER

Local Reaction

At operative site particularly in upper abdomen (first 48 hours)

Treat by skillful neglect

Metabolic Causes

Dehydration acidosis electrolyte imbalance thyroid crisis or hyperglycemia

Initiate intravenous drip of sodium chloride lactate Ringers solution substitute 5 per cent dextrose and add insulin if necessary

Technical Causes

Due to pyrogen in intravenous fluids transfusion reaction (p 319) drug fever (p 94)

Stop all treatment temporarily substitute penicillin for sulfonamide if necessary

Pyuria (p 235^o)

Force fluids and give sulfadiazine with bicarbonate of soda (p 88)

Thrombophlebitis (p 1123)

Usually in leg veins

Heparinize (p 1050) by subcutaneous deposit prepare to ligate saphenous or femoral vein

Wound Infection

After 72 hours

See p 4006

Pulmonary Complications

See p 4016

remove accumulated irritating material from the stomach. Many experienced surgical nurses have great confidence in the efficacy of small sips of carbonated water tea broth ginger ale champagne or essence of pepper mint.

If postoperative vomiting persists or recurs the practitioner should entertain the suspicion that he is dealing with drug idiosyncrasy to morphine demerol or sulfonamide. Drug vomiting will not necessarily disappear when the use of the remedy is discontinued; it may persist for several hours.

Vomiting which begins after the fourth or sixth postoperative hour or which persists beyond that time suggests the possibility of some more serious underlying disturbance such as gastric dilatation adynamic ileus.

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In elderly patients especially those with known renal damage the onset of hiccough at once raises the suspicion of *nitrogen retention* and *impending uremia*. The establishment of the correct diagnosis at this stage permits early treatment of the uremia (p 2276).

Adynamic Ileus—With adynamic ileus there is marked gastric and intestinal dilatation. The condition is suspected when abdominal distention and vomiting continue for more than forty eight to seventy two hours after operation.

The *clinical manifestations* of paralytic ileus are easily recognized. The abdomen is tense. Little or no flatus is passed. On auscultation of the abdomen no sounds are heard indicating that intestinal peristalsis is minimal or absent. There is usually no pain. Discomfort due to distention may be present. Breathing often is difficult due to elevation of the diaphragm. Temperature is normal as a rule but may be elevated if dehydration or infection ensues. Heart action is poor due to pressure upon the heart by the elevated diaphragm and to diminished cardiac output. The pulse is rapid and often thready and weak. Blood pressure usually is low.

As a result of loss of peristaltic power the intestinal canal and stomach are filled with gas and fluid composed of gastric intestinal pancreatic and biliary secretions.

Widespread chemical changes accompany ileus qualitatively those of shock. As the result of the outpouring of gastric secretions into the stomach the *blood chlorides* diminish *carbon dioxide* and *nitrogen* are diffused from the blood into the lumen of the bowel the *carbon dioxide combining power of the blood increases* and the *nonprotein nitrogen of the blood rises*. Thus the patient with severe adynamic ileus suffers from a profound alteration of body chemistry as well as from mechanical disturbance of the cardiac and respiratory systems. Uncorrected the condition often leads to death.

See *Differential Diagnosis of Tympanites* (p 1878).

The active treatment of adynamic ileus is directed towards (1) *decompressing the bowel* (2) *combating dehydration and loss of the electrolytes* (3) *attempts to restore normal gastro intestinal peristalsis*.

Decompression of Bowel—Decompression is accomplished by means of the *indwelling nasal catheter*. The latter is lubricated with *albolene* and passed through one of the nostrils into the stomach. The end of the catheter is attached to a suction or siphon device. If one of the common types of suction apparatus is not available the nasal tube is connected to a piece of rubber tubing sufficiently long to reach to a large bottle placed upon the floor near the bedside. This produces a *continuous siphonage*. If the flow of gastric contents is impeded as a result of plugging of the lumen of the tube suction is applied with a 50 cc syringe. If this fails to overcome the obstruction the injection of 10 to 20 cc of water will reestablish the lumen at once.

A careful daily record is kept of the amount of *gastric drainage*. While the indwelling nasal catheter is in place the patient is given sufficient fluid by mouth to keep the mouth and tongue moist. This does not increase the gastric distention since the fluid at once drains out through the indwelling nasal catheter. A careful record of the fluid intake by mouth

is kept. In computing the actual gastro intestinal drainage this figure is deducted from the total drainage through the indwelling nasal catheter.

In addition to permitting a limited amount of fluid by mouth the patient is urged to *chew gum* several times daily. This keeps the salivary glands active and acts as a prophylactic against acute parotitis which occurs not infrequently when intake by mouth is restricted.

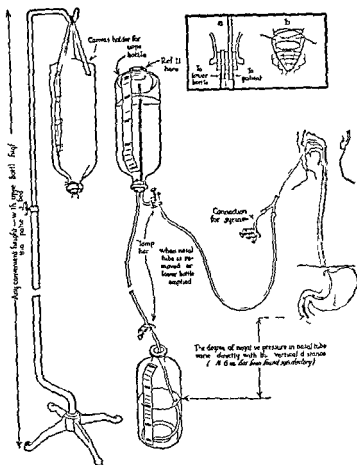


Fig 11 2.—Diagram of suction apparatus employed in the treatment of intestinal distention. An effort should always be made to have the end of the catheter reach beyond the duodenum. Perforations are made so that suction may be extended simultaneously or the stomach and on the duodenum. When the fluid aspirations are great a third bottle should be used as a collecting receptacle.*

Simultaneously with gastric decompression the intestinal tract is emptied from below. This is accomplished by mechanical flushing of the lower bowel. If the adynamic ileus is not secondary to mechanical obstruction or peritonitis the peristaltic stimulants are administered.

The measures used to increase peristalsis are the application of warm packs or turpentine stupes to the abdomen and the administration of

neostigmine 1 cc of 1 2000 solution intramuscularly every four hours After the neostigmine has been injected the patient is given a warm soap suds enema or colonic irrigation

Combating Dehydration and Electrolyte Imbalance—Fluid loss and electrolyte imbalance are corrected by the use of an *intravenous infusion* of 5 per cent dextrose in normal saline or Ringer solution If given continuously day and night at the rate of 30 to 40 drops per minute the total fluid intake varies from 2500 to 3500 cc in twenty four hours After the first liter of fluid containing saline is administered the remainder of the daily infusion is given with 5 per cent dextrose in distilled water to prevent *hyperchloremia* (p 732) A sufficient quantity of fluid is given to produce a urinary output of 1500 cc daily

Restoration of Peristalsis—Efforts to produce resumption of normal peristalsis while simple are of paramount importance The patient must be encouraged to restore the intestinal gradient by the ingestion of warm fluids at the earliest possible moment Soft food is then given Persistent efforts at feeding should not be impeded by nausea or vomiting

The criteria for discontinuing special treatment for adynamic ileus are reduction of abdominal distention free passage of flatus marked diminution of drainage through the indwelling nasal catheter

When drainage through the indwelling nasal tube diminishes the tube is clamped off for two hours During this period the patient is given fluids by mouth A record of the fluid intake is kept At the end of the two hour period the nasal tube is unclamped and permitted to drain freely If relatively little fluid drains from the tube it is an indication that peristalsis has returned and that fluid which has entered the stomach is being passed on into the duodenum If the amount of gastric drainage almost equals or exceeds the amount of fluid which has been taken by mouth gastric peristalsis is still impaired In such circumstances the tube is left in place until such time as the amount of drainage is less than the intake by mouth

Prophylactic Chemotherapy—In conjunction with other efforts prophylactic chemotherapy is imperative to avert the possibility of superimposed infection For wide coverage both penicillin (p 106) and streptomycin (p 103) are advised by intramuscular injection using daily dose respectively of 1 000 000 and 3 000 000 units

Acute Gastric Dilatation—Acute gastric dilatation is a disorder of uncertain etiology Obstruction occurs in the *terminal portion of the duodenum* from pressure of a dilated stomach or constriction by the root of the mesentery Its immediate result is to produce dilatation of the stomach and duodenum proximal to the point of obstruction As a result there is a damming back of gastric and duodenal secretions Since these secretions contain water and inorganic salts which cannot be absorbed in the stomach or duodenum above the point of obstruction *dehydration and electrolyte imbalance* (p 597) quickly develop

The symptoms of acute gastric dilatation consist of *persistent vomiting* of the so called overflow type The patient regurgitates frequently and brings up small amounts of fluid at a time This vomiting is usually unassociated with nausea and usually is effortless The vomitus is dark brown or black in color and is usually foul smelling Distention begins in the

epigastrium and as the stomach dilates tends to become diffuse Discomfort rather than pain is present A succussion splash may be present

As the diaphragm becomes elevated movement of the bases of the lungs becomes restricted and breathing often becomes difficult Such a situation not infrequently sets the stage for the development of *hypostatic congestion* and *bronchopneumonia*

If gastric dilatation is permitted to go untreated the patient's respirations become shallow and labored and the pulse is thready and rapid Fever develops and death in coma not infrequently results The *blood findings* are identical with those already described in *adynamic ileus* (p 4010) They consist of elevated blood carbon dioxide combining power elevated nonprotein nitrogen and lowered blood chlorides

The *treatment* of acute gastric dilatation is essentially the same as that of adynamic ileus (p 4010) It consists of the use of the indwelling nasal catheter to empty the stomach and the replacement of fluid and electrolytes by the *intravenous method* A further objective is to relieve the duodenal obstruction This is accomplished by placing the patient upon the abdomen and elevating the foot of the bed on 18 to 24 inch blocks This maneuver tends to overcome the downward displacement of the stomach and corrects the duodenal angulation

Intestinal Obstruction—Postoperative intestinal obstruction is usually due to external pressure or angulation of the bowel by partially organized fibrinous exudate

The process of healing of operative wounds of the viscera and abdominal wall is accompanied by local fibrinous exudation which tends to agglutinate these tissues to other structures in their immediate neighborhood Under certain circumstances this soft exudate becomes firm and unyielding as the result of organization and resistant bands or veils compress the lumen of the bowel Depending upon the completeness of the compression or angulation the obstruction may be *partial* or *complete* If at the same time the mesentery is compressed the blood supply of a portion of the bowel may be seriously compromised and gangrene threatens

In addition to obstruction by plastic intraperitoneal exudates the syndrome may be a complication of *wound disruption* (p 4006) In such cases a loop of bowel prolapses and becomes compressed between the edges of the wound

Postoperative intestinal obstruction also follows operations upon the bowel The lumen of the gut may be markedly narrowed as the result of *improper placement of sutures*

Symptoms—The symptom of intestinal obstruction due to plastic exudates occur about seven to ten days after operation at a time when exudates become organized and firm If due to wound disruption they may occur at any time If due to the narrowing of the bowel as the result of improper suturing they usually appear promptly after operation

The symptoms of intestinal obstruction consist of nausea and vomiting abdominal distention and increasing difficulty in expelling feces and flatus ending in complete obstipation and inability to pass any gas whatsoever even with the aid of drugs and irrigations

Unlike dynamic ileus organic intestinal obstruction is associated with *cramplike colicky pains* usually located near the site of obstruction

neostigmine, 1 cc of 1:2000 solution intramuscularly every four hours. After the neostigmine has been injected the patient is given a warm soap-suds enema or colonic irrigation.

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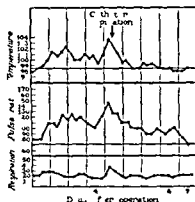
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cases. If treatment is instituted at an early stage before gross suppuration occurs the local inflammatory reaction may be aborted. With frank abscess formation *surgical drainage* is instituted by the specialist.

COMPLICATIONS INVOLVING THE RESPIRATORY SYSTEM

Pulmonary complications are frequent following thoracic or abdominal incision regardless of the anesthetic used. They are largely secondary to *diminished respiratory exchange* and a *diminished volume of tidal air*.



117 —Postoperative atelectasis. *A* Before and *B* after endobronchial aspiration. In the chart above the effect of treatment on temperature, pulse and respiration is evident.

Many factors combine in causing diminished pulmonary ventilation. These include the following:

1. There is *reflex splinting* of the thoracic cage after thoracic operations of the diaphragm and abdominal muscles following abdominal procedures.
2. Postoperative *sedation* abolishes or seriously weakens the cough reflex and depresses respiration.

Symptoms—Parotitis is manifested by swelling and tenderness over the cheek immediately in front of the ear. As the infection spreads the lowermost portion of the gland may be involved. Systemic manifestations include elevation of temperature to 103° or 104° F and rather marked prostration. The pulse rate is elevated and leukocytosis is present. In the later stages suppuration and abscess formation within the gland may occur.

Diagnosis—In the early stage the diagnosis usually can be made in the following manner. A tongue depressor is held by an assistant inside the cheek in order to expose the opening of Stensen's duct. The examiner

TABLE 25^a—POSTOPERATIVE PULMONARY COMPLICATIONS

| | Diffuse Atelectasis | Massive Atelectasis | Pneumonitis | Putrid Abscess | Emboli |
|----------------|---|--|------------------------------------|--|---|
| Onset | Gradual | Acute | Gradual | After 10 days | Acute |
| Chest Pain | Slight | Sharp | Slight | Slight | Sharp |
| Chill | Rare | Frequent | Rare | Rare | Occasional |
| Cyanosis | No | Deep | No | No | Variable |
| Fever | Low grade | Sudden Rise | Low grade | Delayed | Sudden rise |
| Cough | Slight | Sudden | Persistent | Persistent | Persistent |
| Dyspnea | Slight | Severe | Slight | Slight | Moderate |
| Sputum | Slight | Slight | Purulent | Putrid | Blood tinged |
| Physical Signs | Scattered rales | Immobile chest with trachea drawn ipsi laterally | Scattered rales and breath changes | Local tenderness slight dullness | Scattered rales plebitis in calf |
| Radiograph | Diffuse infiltrations | Collapsed lobe | Diffuse infiltrations | Localized cavitation | Diffuse infiltration |
| Leukocytosis | Slight | Slight | Moderate | Moderate | Slight |
| Treatment | Roll from side to side in hale CO ₂ consider prophylactic chemotherapy | As atelectasis but consider bronchoscopy | Chemotherapy if moderate or severe | Surgical drainage Chemotherapy with penicillin | Ligate saphe nous or fem oral veins and heparinize oxygen |

steadies the chin with one hand and gently milks the region of the parotid gland from the area immediately in front of the ear downward and forward. This tends to force secretion from the parotid gland into Stensen's duct. The orifice of the duct is kept in view and soon a drop of secretion is seen. A smear of this material usually reveals the presence of organisms most commonly *Staphylococcus aureus*.

Treatment—Treatment consists of the local application of wet dressings and the prompt administration of penicillin. Systematic local massage carried out in the manner described at intervals of three to four hours is of great value. Irradiation is recommended in more severe

oxygen and 5 per cent carbon dioxide or by having him rebreathe in a bag (4) The patient should be *urged to cough* occasionally especially if there is an indication that there is mucus present in the trachea or larger bronchi. He should be told that although this procedure is painful he cannot harm his incision. The hand is placed on the dressing during coughs to diminish the pain. (5) Early ambulation (p 4122)

Clinical Manifestations.—The postoperative respiratory complications include diffuse and massive atelectasis, pneumonitis, putrid abscess and embolizations. These conditions are considered in greater detail in the chapter on the Respiratory Diseases (p 2013). For present purposes they are summarized in Table 252.

Treatment.—Irrespective of the nature of the respiratory complication prophylactic therapy must be promptly initiated with penicillin using 50 000 to 100 000 units intramuscularly every two to four hours.

OTHER POSTOPERATIVE COMPLICATIONS

Beside the complications encountered in the wound, the respiratory and digestive systems, difficulties may arise in the urinary passages, the circulatory structures and the central nervous system as indicated in Table 253.

3 Pain in the region of the incision puts further restraint on deep respiration and coughing

4 The supine position impairs respiratory motions

5 General anesthetics to a varying degree cause increased bronchial secretion of mucus

The interaction of these phenomena leads to plugging of minute bronchioles and larger bronchi with mucus. Secondary focal areas of *atelectasis* result. On the day after operation auscultation of the bases reveals atelec

TABLE 253—ACTIVE TREATMENT OF OTHER POSTOPERATIVE COMPLICATIONS

Retention of Urine (p 2264)

Give doryl (p 3874) 1 cc intramuscularly and repeat in 2 hours. If ineffectual catheterize after initiating chemotherapy with sulfonamide. If catheter cannot be passed prepare for suprapubic cystostomy.

Anuria (p 2232)

Treat shock if present. Investigate possibility of ureteral ligation or urolithiasis from sulfonamide. Discontinue all medication and institute intravenous drip of 5 per cent dextrose in saline or distilled water.

Urinary Infection (p 2206)

Start chemotherapy with sulfonamide or streptomycin but assure daily urinary output of at least 1500 cc by intravenous drip if necessary.

Backward Failure (p 911)

Stop intravenous fluids. Digitalize cautiously. Give mercurial diuretic (p 2201) if digitalization is ineffectual. Initiate oxygen therapy. Sedate with barbiturate, opiate or demerol. Consider phlebotomy.

Edema (p 706)

Stop intravenous fluids. Give mercurial diuretic if necessary.

Cardiac Arrhythmia (p 873)

Avoid digitalis. Give quinidine sulfate 0.5 gm (7 grains) every 2 hours if necessary. Sedate heavily with barbiturate, opiate or demerol.

Thrombophlebitis (p 1123)

Prepare for ligation of femoral vein and heparinization (p 1010).

Psychoses (p 1376)

Stop all medication. Insist on constant nursing care. Try intravenous paraldehyde or pentothal sodium. Try scopolamine 0.4 mg ($\frac{1}{150}$ grains).

Conalescence

See p 4117

tatic rates in almost every patient who has had an intra abdominal operation.

Measures which promote pulmonary drainage and increase pulmonary aeration help prevent postoperative pulmonary infection. These measures are (1) *Head down position* until the cough reflex returns to promote bronchial drainage. (2) *frequent changes of position* since the dependent portions of the lungs have less pulmonary exchange than the elevated portions. (3) instruction of the patient to take *deep breaths* at frequent intervals. Deep respirations are aided by having the patient breathe air with a *high carbon dioxide concentration* from a tank containing 95 per cent

SECTION XXV
PROGNOSIS

185 Prognosis 4021-4035

CHAPTER 185

PROGNOSIS

HOWEVER much the physician becomes engrossed with problems of diagnosis and treatment prognosis is the main concern of the patient whose first questions—Is the condition serious? Shall I get well? How long shall I be ill? Shall I be able to work again? Shall I be normal again?

How soon may I go home? and How long must I stay in bed?—usually pertain to the outcome. The responses to these questions constitute the art of prognosis.

Upon the ability to master the technics of prognosis will frequently depend the confidence of the patient in the physician and the extent of the physician's practice. The difficulties inherent in the rendition of a prognosis are best understood and appreciated by cataloguing the ingredients that enter into the simplest prognostication. They include

- 1 Knowledge of the nature of the disturbed process (diagnosis)
- 2 Estimation of the natural or spontaneous course of the derangement
- 3 The frequency of complications and sequels
- 4 The effects of the available therapeutic procedures
- 5 The variables relative to the technical skill of the cooperating specialists particularly the surgeon
- 6 Modifications due to nonmedical factors such as the attitude of the patient
- 7 Economic aspects

Not until all these facts are known can the practitioner accurately make his prognosis since a disturbance or dislocation of any one factor may upset all others. The art of prognostication is a prime accomplishment of the competent practitioner. It is a discipline that cannot be learned by rote; it is achieved by long and patient observation and its tempered enunciation requires prior assimilation and adjudication of many facts by a trained and retentive mind.

FACTORS INFLUENCING PROGNOSIS

Nature and Course of the Disease—When the diagnosis has been established the spontaneous course of the disturbance is projected. It may range between self-limited diseases invariably tending toward recovery and those that are utterly hopeless. Between the extremes are the relatively benign and the relatively malignant states.

Benign Conditions—Unqualifiedly benign medical conditions are rare; they include certain of the exanthems such as chickenpox and some of the dermatoses such as pityriasis rosea and acne.

Malignant Conditions—The hopelessly malignant diseases include metastatic cancer and leukemia.

Relatively Benign Conditions—The relatively benign conditions are frequently encountered and embrace systemic abnormalities such as the

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FACTORS INFLUENCING PROGNOSIS

Nature and Course of the Disease—When the diagnosis has been established the spontaneous course of the disturbance is projected. It may range between self-limited diseases invariably tending toward recovery and those that are utterly hopeless. Between the extremes are the relatively benign and the relatively malignant states.

Benign Conditions—Unqualifiedly benign medical conditions are rare; they include certain of the exanthems such as chickenpox and some of the dermatoses such as pityriasis rosea and acne.

Malignant Conditions—The hopelessly malignant diseases include metastatic cancer and leukemia.

Relatively Benign Conditions—The relatively benign conditions are frequently encountered and embrace systemic abnormalities such as the

common upper respiratory infections and local processes like conjunctivitis or urethritis

In prognosticating relatively benign disturbances the practitioner is justified in tempering his reassurance by the warning of *complications*. For example upper respiratory infection may spread to the middle ear or it may descend into the lower respiratory passages under which circumstances a certain risk is involved. If these sequels arise in the face of a completely optimistic prognosis the patient cannot be criticized if he entertains the view that his physician has been careless and short sighted or that more adequate therapy might have prevented trouble.

Relatively Malignant Conditions—Prognostication of relatively malignant disease stresses the factor of *hope*. An occasional recovery may be fortuitously observed from relatively malignant conditions such as miliary tuberculosis or the tuberculous pneumonias.

The advent of a great medical discovery if timely shifts the prognosis from one that is utterly or relatively malignant to one that is relatively or completely benign. In recent times such experiences have been noted in diabetes from the discovery of *insulin* and in pernicious anemia following the introduction of *liver extract*. Who shall say that the future does not hold a similar happy experience for the leukemias?

Complications and Sequels—The complete prognosis takes into account the immediate the distant and the remotely distant eventualities.

The *immediate prognosis* is based upon the course of the uncomplicated clinical disturbance. *distant prognosis* takes into consideration the possibility of complications. Thus acute otitis media is a relatively benign process provided that it does not become complicated by mastoiditis or vein infection. The prognosis for the otitis is excellent but that of the complicating mastoiditis may be serious or even grave. A local pyoderm such as a furuncle or a carbuncle is not prognostically serious so far as the local skin and cellular tissues are concerned spread of the infection with involvement of venous channels bacteremia and pyemia however shifts the prognostic scales from the favorable to the serious side.

The *remote distant prognosis* is concerned with late sequels. An acute attack of rheumatic fever is rarely fatal and often pursues a misleadingly benign course. The complicating valvulitis which may not become apparent for many years produces cardiac invalidism and most of the cardiac fatalities in the young and middle aged.

The long range view of a necessity deals with *recurrences remissions and exacerbations*. The prognosis for an isolated period of depression in the manic depressive is excellent but there is a better than even chance that the episode will recur. gastroduodenal ulceration usually responds to therapy but recurrence is the rule and a similar situation prevails in rheumatic fever. the immediate postoperative results in herniotomy are excellent but over a period of longer follow up recurrences may approach or exceed 10 per cent. renal calculi often pass spontaneously but the formation of later concretions is the rule rather than the exception.

In *recurring disturbances* the prognosis deals with the immediate attack and the future course. Both short range and long range views are to be communicated to the patient. An interim program aimed at the prevention of recurrence is outlined. The patient is instructed as to early pre-

monitory symptoms so that no time is lost in instituting active treatment. Frequent follow up visits in health are suggested since objective findings may precede subjective complaints. A notation on the desk calendar assists the physician to summon his more casual and forgetful patients.

Therapy and the Therapist—The prognosis of disease is altered by the potential accomplishment of the therapist. How different has been the medical outlook in syphilis before arsenicals, in pneumonia and streptococcal infection without penicillin, in pernicious anemia without liver extract, in acute appendicitis without surgical therapy, in diphtheria and tetanus infections without the specific serums.

The human equation in treatment greatly modifies the outlook. Many technical procedures require apparatus and a skillful technician; the results in irradiation therapy of malignancy depend in great part upon the power of the apparatus that is available; the removal of an endobronchial tumor or a foreign body requires both a bronchoscope and a skilled bronchoscopist; operative risks are dependent upon the aptitude of the individual surgeon; appendicitis mortalities vary appreciably in different institutions and on different surgical services in the same institution. Experience is a determining factor in surgical prognosis; an operator will have many more complications in his first hundred thyroidectomies than in later groups.

The Attitude of the Patient—The attitude of the patient toward his disease is of great prognostic significance in the protracted and chronic diseases. Individuals vary between those who have an indomitable will to live and those who display little or no fighting spirit. One of the great privileges of general practice is to witness the spectacle of the patient who simply will not be defeated by his infirmity. The greatness of the human spirit with its courage and heroism can never be better exemplified than in the conquest of chronic disease such as tuberculosis or crippling arthritis.

At the other extreme of human behavior are those who willingly or even avidly accept invalidism and death. Weaklings and the readily vanquished find a solution for an unsuccessful career with a spot on the lung, a touch of rheumatism, spells and fits, high blood pressure, or a heart attack. They capitalize infirmities by collecting various types of insurance; they bully the circle of relatives and acquaintances by the strength of weakness.

The attitudes of relatives and friends may determine in which group the patient is to gravitate. Each practitioner of experience recalls the heroic mother who simply will not permit her child to become invalided or succumb. A young tuberculous patient may be nursed to health by faith or an abiding love.

The character of the physician also influences the reaction of his patient. Excessive pessimism, a sense of defeat or an unconvincing hilarity may completely unnerve the unstable patient; the overhearing of careless remarks or misinterpretation of medical conversation may plunge a patient into the depths of despair.

On the brighter side the determined physician may carry his patient along through years of chronic illness and infirmity. Facing a difficult situation with a sense of reality and invincibility, he may sweep his patient along with him toward health; he may teach the use of limited resources to the end that life again becomes vivid and interesting.

Social and Economic Factors—The prognosis of disease may depend upon the extraneous variables such as the understanding and intelligence of the patient and his economic ability to procure proper treatment. It is difficult to teach dietotherapy to ignorant illiterate patients suffering for example from diabetes. An awkward and clumsy individual cannot be trusted to self administer hypodermics of insulin.

Treatment of the Haves and Have Nots—Economics plays an important role in therapy though not always in the manner that may be superficially suspected. There are times when excessive opulence as well as excessive poverty defeat the therapist. With the assistance of social agencies and endowed or public institutions the resourceful practitioner usually finds a way of overcoming economic obstacles so as to obtain adequate therapy for the poor and underprivileged. Dealing with the excessively opulent is much more difficult. These patients often think that their wealth entitles them to buy a cure or relief of a medical condition. Physicians and surgeons are hired and fired in the manner of domestic servants and the uncertainty of his tenure undermines the loyalty of the practitioner. The privileged not only suffer from charlatanism but their medical infirmities are weighted down by cooks and bartenders. The physician finds himself struggling against the pathological condition plus the fleshpots.

The wealthy wage earner is a most trying patient. He has a multiplicity of responsibilities. It is difficult for him adequately to care for his own health and welfare yet he donates money to institutions in order that underprivileged patients may receive the rest and sanctuary that he owes to himself. Least grateful of all are those patients who have not earned their advantages. Ease has made them soft in mind and body and a therapeutic regimen is subject to their whims. In their care the physician must often conclude that his efforts are being squandered.

The Tangibles versus the Intangibles in Prognosis—Many attempts have been made to standardize prognosis by clinical aphorisms and rules. In the early days of the stethoscope the prognosis was often determined by the murmurs that were heard. With the introduction of the sphygmomanometer prognostic rules were based on the height of the mercury column. More recently judgments are given on the electrocardiographic tracing. The physician of today recognizes the folly of rendering a prognosis based for example on murmurs. He is learning that it is equally fallacious to utter judgments based on a blood pressure reading and he will shortly learn that the same is true of an electrocardiographic tracing.

A common cause for error in prognosis is the failure to recognize that patients rarely die of organic disease. They usually succumb to a breakdown of a physiological mechanism that is often immeasurable. The physician of experience recognizes when his patient is seriously ill and has little need of instrumental or laboratory corroboration. Indeed his clinical judgment may be diametrically opposed to the objective findings and the laboratory report may be quite favorable when the clinical aspects are ominous. A normal leukocyte count for instance is frequently associated with gangrenous appendicitis. On the other hand unfavorable laboratory and objective findings may be offset by clinical appearances that are favorable. A marked hypertension and an evil appearing electrocardiogram may be

present and persistent for many years and even decades in patients free from subjective symptoms so long as they are not given a profound anxiety or panic state. Each practitioner has in his practice many patients who have shown old tuberculous scars and even cavities for innumerable years; others live the normal span of life with various types of cardiac murmur; still more manage to carry out a full existence despite positive serologic evidence of syphilis.

The practitioner cannot, however, wave aside lightly an ominous objective or laboratory finding. In the face of good function he may justifiably prognosticate from physiology and hope rather than from pathology and despair. While this attitude may evoke chortles from the autopsy



Fig. 1176—Chest film of E.J.A. (who accompanied Dr. E. L. Trudeau to the Adirondacks) fifty-seven years before his death. Except for two years spent with Dr. Trudeau in founding the Sanatorium, he paid no heed to his pulmonary condition and led a full and active life. Film reveals a huge cavity at the right apex, calcific infiltration at the right upper lobe, and fluoroscopic findings suggesting an adhesive pericarditis, probably tuberculous. Additionally, physical examination showed a mass in the right upper quadrant, presumably a tuberculous perisplenitis. At no time did the patient suffer from his infection, and he died of progressive circulatory failure at the age of 78.

room, the policy permits a large number of pathologically doomed patients to enjoy considerable physiological pleasure and happiness for surprisingly long periods of time (Figs. 1176, 1177, 1178, and 1179).

Statistics and Prognosis.—In individual prognosis, statistics function as a weather vane. From them the practitioner recognizes the wind direction; he knows nothing of wind velocity or of weather conditions such as temperature, humidity, or visibility.

In a tangible disease such as pneumonia, statistics give an overall mortality. However, the outcome in pneumonia is dependent upon many variables, including the age of the patient, the type of pneumococcus, the general nutrition of the sufferer, and the promptness and completeness with which therapy was initiated and accomplished. How is it possible for there

to be statistical evidence concerning a Type III pneumonia in a 40 year old laborer moderately alcoholic with three lobe involvement an old tuberculosis a tendency toward glycosuria and ill forty eight hours before treatment could be initiated? It is doubtful whether the mathematical

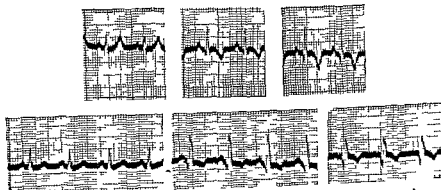
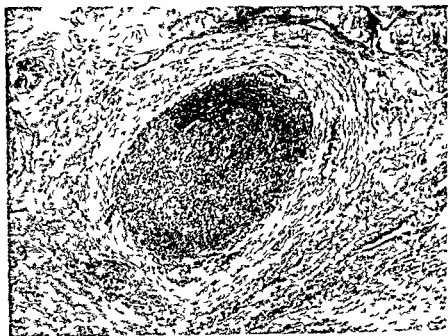


Fig 1177—Upper figure Section through artery showing characteristic changes of thrombo-angitis obliterans (November 1932) Upper electrocardiographic trace (January 1932) shows features typical of coronary closure presumably also on the basis of a thrombo-angitis Lower electrocardiographic trace (April 1943) eleven years later shows a little significant change despite the fact that the patient has led a full and demanding existence and has continued to smoke moderately

geniuses from Newton to Einstein could work out the possible risk even if all of the data were available

Actuaries Life Insurance and Prognosis—The life insurance companies measure human life and its span by tangible yardsticks these include height weight age pulse rate blood pressure urinalyses electrocardiograms and any objective laboratory test that can be seized upon as a prog

nostic index. The average figures and the average experiences become the criteria by which the viability of the individual patient is estimated.

The practitioner often finds himself in conflict with the life insurance investigators and their medical staffs. He has the frequent experience of having perfectly healthy specimens rejected for life insurance because of some individual variation that is within the limit of normality. On the other hand, occasionally those whom he regards as bad risks are accepted without qualification.

The acceptance or non acceptance of risk is the business of the life insurance company and its medical department. The practitioner's problem is that of dealing with the insecurity of the patient who has been refused insurance. Under these circumstances the patient may conclude that his practitioner is not competent to diagnose medical infirmities, that his physician has withheld ominous information from him, or even that if his present condition is favorable, he faces some type of impending disaster in the near or distant future.

In any event, equilibrium is disturbed. Confidence and faith in the physician are undermined through the stupidity of attempting to express an art in terms of mathematics.

The Individual Prognosis—Prognosis is distinctly an individual matter. It is an art and not a science though it deals with certain tangible information.

The practitioner has no function more important than that of attempting a prognostic evaluation. While his prognosis must be sufficiently definite to be satisfying, it should also be sufficiently explanatory so that the patient and his relatives recognize the necessary limitations of prognostication and the possible variables that may modify the picture.

To be an expert prognostician, the practitioner has only to combine the virtues of a scientist, a pedagogue, a public relations counsel, and an oracle.

THE TYPES OF PROGNOSIS

The prognosis may be favorable, guarded or unfavorable, or immediately or remotely fatal.

The Favorable Prognosis—There should be no hesitation or reticence in rendering a favorable prognosis. This is of particular importance when the anxiety of the patient verges on the panic state concerning what is medically a trivial condition. Thus a mother with her first child may become hysterical because her child has swallowed a bottle of ink, or because the temperature has risen to 103° or 104° with a slight chilliness accompanied by some cyanosis. Under such circumstances the distraught woman has no interest in the diagnostic problem, but wants simply to be told that the child is in no danger. It is quite justifiable to gloss over any uncertainty in order to lend security.

Many adults live in fear of cardiovascular accidents, such as cerebral apoplexy and coronary thrombosis. They believe that each headache or chest pain is indicative of an impending disaster and come to the physician for prognostic soothing rather than a prescription.

The practitioner should unhesitatingly herald his prognosis when it is unqualifiedly favorable. The discussion concerning the diagnosis and the

variants may well wait until reassurance has poured its oil on the troubled waters

The Guarded or Unfavorable Prognosis—When possible the guarded or unfavorable prognosis should be tempered by emphasis and if necessary exaggeration of the favorable and hopeful aspects. The ominous qualifications may be added later and correctly adjudicated. There is no exaggerating the favorable therapeutic value of hope and the unfavorable influences of fear and despair. The practitioner must often sacrifice his reputation as a prognostician in favor of the humanities.

In cardiovascular renal diseases the cardiologist is not infrequently beset with shibboleths of evil prognostic significance based upon murmurs electrocardiographic tracings or sphygmomanometric readings yet the patient with advanced hypertension may survive twenty or thirty years only to die of a disease totally unrelated to the vascular disturbance. Again after a cerebral vascular accident with hemiplegia a patient with marked hypertension may live for decades patients with murmurs due to well developed valvular defects may lead useful and active lives and reach old age without any mishap the presence of coronary occlusion may be but is not necessarily of ominous prognostic import, heart block may be pres

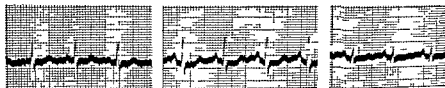


Fig 1178—Electrocardiographic trace of a 73-year-old woman. Shows relative normality despite known hypertension for more than twenty years. There is no evidence of cardiac enlargement there are no fundus changes and no albuminuria—despite the fact that for the last ten years the systolic tension has never been less than 190 and often exceeds 230 and the diastolic tension has never been below 100 and was more often between 110 and 120.

ent for many years without producing serious disability. Longevity is quite possible also in patients with fibroid phthisis, chronic lymphatic leukemia, renal insufficiency or an advanced diabetes mellitus.

The apprehension aroused by solemn utterances frequently causes more damage than the disease process. It gives rise to an aura of anxiety symptoms while bland reassurance may allay anxiety and permit the patient to survive comfortably in the presence of advanced pathological change. The extravagant therapeutic claims of ignorant charlatans and the denial of disease by the faith healers may produce similar happy though superficial psychotherapeutic effects to the temporary chagrin and irritation of the more learned but less astute physician.

The Immediate Fatal Prognosis—The physician must reserve the immediate fatal prognosis until every bit of evidence is irrefutable and unchangeable. He must assume that his patient will survive as long as he has a scrap of ammunition in his therapeutic armamentarium.

Every effort must be made to withhold knowledge of the fatal prognosis from the patient. The practitioner must lie to him and if necessary swear by all that is sacred that he has not abandoned hope. The nearest of kin or a close friend should however be told the whole and complete

truth for the protection of patient and physician. Exceptions to this generality exist in the case of Catholics who desire to have the last rites of the Church and men of property who have not put their affairs in order. Even these may be comforted by the statement that the religious or legal technicality is being executed because of a remote chance of fatality; the patient will rarely be fooled by this while he but it is well worth the effort.

Vigorous efforts to prolong the existence of a doomed patient while well intentioned are not always in the best humanitarian tradition. It has been said of this practice that the physician prolongs dying not life. It is cruel to prolong suffering but while the physician cannot and dare not do anything that will shorten life he may cheat death of its horrors by liberal doses of sedative and narcotic.

It is interesting to note the tenacity of life when the dying patient is given nothing but opiate. I have the distinct impression that the stimulated patient dies sooner. This observation is not remarkable when the pharmacology of the allegedly stimulant drugs is considered. Caffeine and

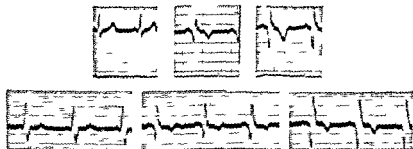


Fig. 11-2.—Upper leads taken after acute coronary thrombosis, October 7, 1929. Lower leads taken fifteen years later (April 17, 1944). Note Q and Q3 deformities of the R-T segment and inversion of T-S. In the fifteen years between first and second tracings the patient led an active life without significant modification of his business activities.

its derivatives, camphor and its substitutes, strychnine injections of epinephrine and other sympathomimetic amines, and overdosage with digitalis bodies, particularly strophanthin, each given by the hunch and whim method would probably defeat a normal mechanism—much more one that is on the losing side of a struggle with a deranged mechanism.

The Remote Fatal Prognosis.—A serious dilemma arises when the physician becomes aware that a fatal issue is to be expected in a patient who is seemingly reasonably well. Thus, advancing hypertension with a rising diastolic pressure, the appearance of retinal hemorrhages and exudates, a fixation of the specific gravity of the urine, and an electrocardiogram that shows changes suggesting progressive myelomalacia are compatible at one time or another with apparent well being. Yet it is obvious to the practitioner that the number is up.

It is a cruel ordeal to be compelled to give the accurate prognosis to the patient yet individuals with responsibilities are entitled to know enough of their prospects so that their affairs can be arranged. They must be told in a kindly and gentle way that a fatal issue is a possibility rather

than a probability or a certainty. They must be given the opportunity of seeking or initiating any type of hopeful therapy while there is still time and opportunity.

THE THERAPEUTIC VALUE OF A FAVORABLE PROGNOSIS

It would be impossible to overstate the therapeutic value of a favorable prognosis. When the facts warrant the optimistic report, there is no problem but even in the face of adverse findings, the physician cannot be criticised for sacrificing his scientific accuracy and integrity for the benediction and blessings of hope.

Very rarely have patients the courage to face truth. In adversity each of us is anxious to practice self-deceit. Under some circumstances the physician deludes his patient with hope as a narcotic, reserving the truth for a friend or a relative. Rarely in dealing with a truly heroic soul, the patient demands to be told the truth and requests that all others be deceived.

Occasionally the practitioner shoulders the entire responsibility and discloses to no one the ominous situation. In so doing, he exposes himself to criticism which is often justified. Once in a great while he enjoys that indescribable satisfaction that arises from having spared another human being from untold anguish.

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APPENDIX

ESTABLISHING AN OFFICE PRACTICE

PREPARATION FOR THE ROLE OF PRACTITIONER

The doctorate in medicine is obtained after an exacting series of accomplishments the degree of Bachelor of Arts or Bachelor of Sciences being a prerequisite for admission to most medical schools. Of college graduates applying for matriculation in the medical courses a small number are admitted for training. The successful matriculants are chosen after careful scrutiny of scholastic standing, personal attributes, campus activity and social, economic and geographic background.

The curriculum of the medical student includes instruction in the *fundamental sciences* (including anatomy, histology, embryology, physiology, biochemistry, pathology, bacteriology, immunology, chemistry and pharmacology) and the *clinical disciplines* (including the practice of medicine, the practice of surgery and the various specialties of laryngology, anesthesiology, otology, gynecology, obstetrics, pediatrics, ophthalmology, urology, orthopedics, radiology, neurology, psychiatry, dermatology, parasitology, gastro-enterology, endocrinology, syphilology, public health administration and nursing).

Of each entering class in the modern medical school not more than 75 to 80 per cent survive to graduation. Thus comparatively few who aspire to a medical career successfully obtain their doctor's degree.

The License to Practice Medicine—The recipient of the academic degree of Doctor of Medicine cannot legally engage in practice until he has passed the examinations of the Boards of State or National Licensure. Increasingly and rightfully these Boards require intern training in a qualified hospital. For the fulfillment of these requirements and in the interest of his own progress and welfare the medical graduate seeks a desirable appointment.

Internship and Residency in Approved Hospitals—The American Medical Association annually publishes the roster of approved hospitals whose equipment and qualifications satisfy the requirements for satisfactory intern training. This roster is made up almost exclusively of (1) *university hospitals* usually affiliated with the medical school, (2) the *privately endowed hospital* often supported by a religious body, (3) the *doctors' hospital* founded and managed by an outstanding physician or group of physicians, and (4) the *municipal, county, State or Federal hospital*.

Choice of Hospital—The more desirable hospitals select interns from hundreds of applicants. In the approved university hospital the intern serves under a staff which is usually identical with that of the medical school. In essence he receives a postgraduate university course; this type of training is best suited for those who plan an *academic life* in medicine.

The neophyte who intends to enter *private practice* finds his require

humility that have yielded much in later years. Often in the fuller growth of maturity they have outstripped those whose good fortune it was to start the medical career with greater advantages.

Specialization and Certification—Those physicians who elect to become specialists and desire national certification in a chosen field are required to have *resident training*. Appointments to residencies in internal medicine, surgery or the specialist fields are open only to the graduate intern.

The medical student who runs the gamut from the undergraduate medical course to the senior residency may spend eight to ten years securing his professional training. Certification by the *National Boards* requires additionally several years of active practice. The young physician will do well to remember that it is becoming increasingly important to prepare for *national certification*. This is becoming a prerequisite to appointment on attending staffs and for approval for insurance compensation and referred practice (p. 3650).

American Boards have been established in Anesthesiology, Dermatology, and Syphilology, Internal Medicine, Neurological Surgery, Obstetrics and Gynecology, Ophthalmology, Orthopedic Surgery, Otolaryngology, Pathology, Pediatrics, Plastic Surgery, Psychiatry and Neurology, Radiology, Surgery and Urology.

THE PRACTITIONER AND THE COMMUNITY

The practitioner's choice of his community is too often a matter of chance. Some physicians sponsored by older relatives or friendly established practitioners enter naturally into their chosen lives and practice in their own community. Others shun the familiar places and seek to practice amongst strangers. All too often the decision is made without the mature judgment that the problem merits. The urgent demands made upon the student first in his undergraduate years and then during the bustle of the internship result in a lack of studied consideration of the details of private practice. It is not until the doors of the institution are virtually closing upon the graduating intern that he ponders his own future.

The lack of guidance in the determination of the locale for private practice has resulted in a concentration of doctors in the cities and a paucity in rural areas. The urban doctor whose expenditures are excessive and income limited struggles for existence with stark realization that his training and scientific resources are being inadequately utilized. Mean time rural communities throughout the country receive sketchy medical care because of limited facilities.

To his community the private practitioner owes gratuitous attendance on the poor and the underprivileged. In the clinics and hospitals as well as in his private office the physician cannot and will not reject a patient because of inability or failure to pay a fee. The physician carries the medical burden and responsibilities of his own family and relatives near and far as well as those of the defeated and unfortunate members of the environs.

Unlike the business man the practitioner attempts to prevent rather than promote those factors which might prove of personal economic advantage since his best efforts are directed toward the reduction of the

ments better met in the private hospital those commanded by older physicians or in the local community or city hospitals where the attending staffs are recruited from men in active practice

The State and Federal hospitals offer their best opportunities to those who aspire to be *career men*. Unusually fine training and an attractive salary are afforded those who desire to enter governmental service

The Intern Training—Intern training is a valuable part of medical education. The intern deals at first hand with clinical material and has the opportunity of studying and caring for patients under optimum conditions. Problems of economics are remote, the hospital administration provides and maintains equipment and supplies, laboratory aids are easily obtained at little or no cost and nurses, orderlies, technicians and secretaries are in attendance. Senior attending physicians assist and advise and consultants are readily available.

Though few physicians realize their blessings during the tenure of the internship, they are probably practicing the science of medicine under more nearly ideal conditions than they will ever again experience.

Doctor-Patient Relationship in the Hospital—Kindly and lucid medical exposition, the recognition of the individual component in disease, knowledge of the complicated interrelationship of the units that make up the family or the community mark the broadly cultured and socially useful doctor of medicine. It is a misfortune that most institutional physicians are too busy or too self-important to enter into explanations to the laity.

During his internship the young doctor has the opportunity of obtaining extracurricular training in *human relationships*. He learns that his patient is an individual rather than a case or number through conversations with the patient, his relatives or friends. The young physician may progress in the study of medical exposition to the laity. It is no easy task to explain a complicated technical situation to a patient who is emotionally involved.

The thoughtful intern discovers that the point of view of the patient is often diametrically opposed to that of the physician. The principal medical interest is often the *establishment of a diagnosis*; the patient's chief concern is *prognosis*. Whatever the naming of the illness, the sufferer wants to know whether he will recover and how long and to what degree he will be incapacitated.

These exercises in the *doctor-patient relationship* serve in good stead in later years of general practice. A citizenry exposed to a deep-rooted doctor-patient relationship will never willingly permit the practice of medicine to become depersonalized.

Practice Without Intern Training—The less fortunate graduate who trains in the less qualified hospital is greatly dependent for his training upon his own resources. Those who are unable to secure an internship and must enter directly into practice lack inestimable advantages. Nevertheless, by careful scrutiny of the medical literature, by attendance at ward rounds, medical meetings, conventions and post-graduate courses, and by close observation and keen study of the clinical material of private practice, splendid proficiency may be achieved in the art and science of medicine.

Many of our finest physicians have been thrown wholly upon their own resources; they developed an independence, an intellectual curiosity and a

world at large *Secret remedies secret cures or trade secrets are not countenanced in medical practice*

THE ECONOMICS OF PRIVATE PRACTICE

The practitioner also owes an obligation to himself and his family. In many instances his training has been accomplished through sacrifices and deprivations by relatives and friends.

Wherever he may locate the neophyte enters a cold world of reality. During his student and institutional life he has been sheltered from the economic stress and strain of human existence. As a practitioner he is faced with the problem of providing food and lodging for himself and his dependents. The new problems involve simple economics and manifold obligations toward the individual patient, the unit formed by family and friends, the community, the local hospital and the medical profession as a whole.

The average practitioner starts life with little or no funds of his own and only casual opportunity for earning money. At the outset of his career he is frequently burdened with debts and obligations. A mature adult—for few men enter practice before their twenty-fifth year—he has the normal desires for a home and a reasonable degree of economic security for marriage and the privilege of rearing his own children. In fact, he is handicapped in his outlook if his own personal life has not included the usual experiences of the average man.

The doctor rarely has resources other than the fees that accrue from medical practice. He cannot resort to advertising or mass production and he obtains no royalties for work done. Moreover, his greatest demands come from the poor and underprivileged members of the community whose medical handicaps further limit their ability to pay an adequate fee.

The physician must run the difficult course of making provision for himself and his family and yet remain within the drastic confines set by the ethics of his profession and by his conscience. Under severe economic stress it is difficult for the practitioner to eschew the easier way that is characterized by exploitation of the patient and splitting of fees.

The majority of private patients expect to pay a reasonable fee for professional services rendered. Nevertheless, a considerable proportion of private practice is gratuitous and includes the poor and indigent, real or simulated, the family and family-in-law, colleagues, nurses, clergymen, pharmacists, social service workers and their immediate cohorts. The practitioner renders free service in his office, in the community hospitals and in clinics. He receives no "retaining" finding or split fee as lawyers sometimes do, nor is he paid preferentially as is the mortician. In fatal cases his bill for fighting the mortal struggle is often less than that of the undertaker who is paid first, and it is usually inconsequential in comparison with the fee of the lawyer who probates the will. Notoriously, the doctor's charge is the last bill paid.

The practitioner in his own interest and for tax purposes must establish a business system. Ledgers must be set up, billings and collections must be arranged, taxes must be calculated and paid. Conscious of the complex difficulties it is no wonder that many seek the shelter that is

incidence and extent of disease. He serves to protect his community by supporting and enforcing all public health measures. He is in his highest development the local pedagogue of hygiene and preventive medicine.

THE PRACTITIONER AND CLINIC OR HOSPITAL

It is virtually impossible for the physician to survive in his community unless he joins the staff of the local clinic or hospital. On a purely practical basis this affiliation is essential since without it he has not the privilege of treating in hospital those private patients who require it. Moreover the hospital appointment serves to keep him abreast of medical progress. He learns in his institutional work of the experiences of his colleagues with new procedures, preparations and techniques. He meets them as he makes his rounds and his medical acumen is refreshed and sharpened both by competition and by exchange of ideas with them.

Unfortunately however appointments to desirable visiting and attending staffs are not always easy to obtain. In many instances their achievement is dependent upon factors other than professional attainment. The extraneous factors of friendship, race and creed often bar a competent applicant and favor one who is incompetent. In municipal and state institutions politics rears its ugly head in the private hospitals which are usually controlled by sectarian lay groups, partisanship and denominationalism may offset medical ability.

Some of the gravest injustices in medical practice result from the attitude of the closed hospital which may bar the competent but politically undesirable physician. Under our present arrangement a physician with adequate credentials on one side of the street may lack hospital privileges on the other side. While no conscientious practitioner would urge the closed hospital to lower standards at the same time there must be some more equitable arrangement for the conscientious man without connections who finds it necessary to hospitalize his patient.

THE PRACTITIONER AND THE MEDICAL PROFESSION

The conscientious practitioner owes loyal support to his profession. The constant growth of the medical sciences requires organizations for the promulgation and discussion of scientific advances, publications for the distribution of recently acquired knowledge, libraries for housing the voluminous medical literature and meeting places for the discussion of current problems and for contact with leaders and workers in the various medical fields.

To support these activities, participate in their benefits, assist in their maintenance and continue his own post graduate training, the practitioner must recognize a continual obligation to his local and state medical societies, the American Medical Association and the specialty boards of local or national scope.

Each practitioner of medicine functions as the sole representative of his profession in observing the clinical material that comes under his scrutiny. Many of the great medical contributions, such as those of Sir James Mackenzie, have come from simple bedside observation. These valuable additions to our knowledge belong to the profession and to the

ment Eventually each successful practitioner will be impelled to practice in his own individual way. The more fortunate may afford the necessary outlay at the outset but others through necessity must serve an apprenticeship rent or share offices until the economic requirements can be met.

In the establishment of the office the first and most important decision is its *sit*e. Should the office be located in the home or is it better to rent space in a professional building? Each plan has advantages and disadvantages.

Conditions of practice vary in different localities. Particularly in small communities professional suites are not available and physicians practice at home. In many larger cities it is the custom for the physician to rent space in a professional building.

The Home Office—The home office is economical in that there is only one rent the housekeeping maintenance presents an extra burden but very little extra expense the practitioner need not travel innumerable times from home to office there need be no set time for his appointments the patient assumes that his physician is available throughout the hours of the day and night.

The *disadvantages* of the home office are both professional and personal. Unless special architectural plans have been drawn to separate home and office there is very little of a professional atmosphere since the combination is almost invariably a makeshift. The patient does not have a sense of privacy and it is difficult to maintain any confidential relationship. The doctor and his family live the existence of the statues in the town square members of the family attempt unnoticed to steal in and out of the house they must maintain silence while patients are being examined during office hours they attempt to be both invisible and inaudible. The physician is deprived of needed relaxation less thoughtful members of the community drop in at odd times and one or two such interruptions of an evening or during the day or on Sunday or a holiday disrupt any opportunity for rest relaxation reading writing or entertainment.

The solution of the home office has been offered by many architects. Homes can be built with a separate wing for professional use. A private entrance for the office separate and apart from the living quarters affords the fortunate practitioner an arrangement that includes all of the advantages and excludes most of the disadvantages of the home office.

Barring this ideal type of arrangement the majority of physicians must improvise for the home office. The country practitioner will use his house the city physician his flat or apartment.

The Office in the Professional Building—The alternative to the home office is an office in a professional building. The physical layout of the professional office is usually more practical than the improvisation at home there is less of the atmosphere of amateurishness and the patient feels that there is more privacy. Against this the professional building smacks more of a commercial arrangement. There is not the same feeling of warmth that is present when the physician is consulted at home the professional office means double rent separate arrangements must be made for housekeeping the practitioner must travel from home to office many times during the course of the day.

On the positive side of the ledger the family enjoys the privacy of a

offered by academic life and salaried positions. Only the braver and more resolute dare the hardships of the private practice of medicine.

PERSONAL PROBLEMS OF THE PRACTITIONER

In addition to his functions in the practice of medicine the practitioner is faced with those responsibilities and problems of every day living which confront every other man. He has a home to establish for instance and children to rear and educate. He has his own extra medical interests to satisfy and hobbies to pursue. There grows in him a great desire to have time for reflection and meditation.

In reality the successful practitioner has but a small amount of borrowed time in which to deal spasmodically with his own economic and personal affairs. Sooner or later all hope is abandoned for a rubber of bridge, a round of golf, a quiet family evening, an uninterrupted session at the movies, the theater or even the radio, or for that heart-to-heart talk with the long suffering wife or junior, or his sister. Vacations, holidays and a bit of sanctuary become mirages.

In addition to the professional attainments and skill the practitioner must heed broader disciplines in his human relationships. His personal habits and way of life are expected to be above reproach. In his morals, his personal habits and his appearance the doctor must be impeccable for scientific ability will not offset questionable morals, alcoholism or physical uncleanness. He must in other words recognize and cultivate certain intangible factors vital to good practice. Robert Louis Stevenson has said that the physician is 'the flower (such as it is) of our civilization.' That distinction is not earned without cost.

THE PRACTITIONER AND HIS OFFICE

The physician may enter general medical practice in one of a variety of ways. The simplest introduction is rental or subrental of an office that is already functioning. The establishment of a personal and individual office is more difficult but much more desirable.

Entering the Established Office—The practitioner may start practice in an established office in one of three ways. He may be the assistant to an older man, he may rent time or space in the office of another physician or he may join a group. None of these arrangements requires significant capital expenditure or burdensome planning. The tyro need bring only his few personal possessions and he is quite ready to receive patients.

The disadvantages of entering the established office are manifold. Essentially there is a submersion of the individual personality. The physician who is introduced as an assistant is apt always to be regarded as an assistant where space is rented or shared the lessee has never the feeling of proprietary right, friction may develop due to the overlapping of time or patients, the office personnel may please one member of the group but not the other. Sooner or later the practitioner who is to be marked for success outgrows the arrangement. He finds he needs more time or more space and it becomes essential for his progress and imperative for his self satisfaction that he establish and maintain his own office.

The Establishment of a New Office—The establishment of a new office requires capital expenditure, planning and purchase of materials and equip-

The Consulting Room—In the home the consulting room is situated in the transformed back parlor or dining room. The consulting room should contain a desk with its chair for the physician and at least three or four other chairs for the patient and his entourage.

A *filing cabinet* for the storage of professional and business records should be close at hand. Since these records contain much personal information the cabinet should be locked at all times. Patients should be prevented from reading their own records and certainly must not be tempted to scan the records of their fellows.

The severity of the office can be relieved by appropriate decorations for the desk and walls. Cheerful prints, etchings and photographs are in expensive and particularly pleasing diplomas and the like are best hung in the examining room.

THE DOCTOR AND HIS TELEPHONE—Perhaps the most important instrument in the office is the telephone which is both the blessing and the scourge of medical practice. The telephone serves great purpose when in an emergency it hastily contacts the physician and his patient. Unfortunately in the vast majority of instances it is the curse of good medical practice. The greater part of the time it links inexorably the harassed practitioner and his least considerate and most demanding patients. To these no time of the day or night seems sacred. The telephone will inevitably ring during the most intimate details of a history in the midst of the pelvic examination during the course of the favorite radio program and at diabolically spaced intervals during the night. If the physician answers his telephone placidly and kindly his telephone practice will increase to the point where he can do little else. If he becomes irritable and vexed his patients regard him as a wicked character.

The only possible solution for the evil of telephone abuse is the attempt by the physician to telephone active patients at some stated hour. If he is successful in contacting them he may enjoy some surcease. The solution however is usually only theoretical since the more obnoxious patients will invariably be 'busy' or out. If they are at home they will forget the most important detail and ring back anyway.

LIBRARY—The doctor's library which best graces the walls of his consulting room consists of medical and nonmedical texts and current journals. The books purchased in medical school are the basis of most collections. Handbooks of therapy are of greatest value for current needs. The *State Journal of Medicine* and the indispensable *Journal of the American Medical Association* are essential for the physician who plans to keep up with the progress of medicine.

Special journals are chosen according to individual bent but more helpful than these publications for the general practitioner are the less formal *Clinics* which deal more succinctly with commoner practical problems. Grateful patients may be told that subscriptions to these make more appropriate gifts than neckties, cigars and desk sets.

One of the great contributions of Sir William Osler was his emphasis upon the nonmedical reading of the physician. The undernoted list has given greatest joy and benefit to the present author. "The Bible Designed to Be Read as Living Literature," "The Jefferson Bible," "The Plays of Shakespeare," "Bacon's Essays," "Autobiography of Benjamin

home the physician's hours of service are more limited since patients are less likely to invade living quarters that are not officially intended for professional use

The Office Units—Whether the office is maintained in the home or in a professional building it must contain at least three basic units the *waiting room* the *consulting room* and the *examining room*. For economy the consulting and examining rooms may be combined so that there are only two rooms from the standpoint of rental. Under these circumstances the examining table is placed in a cubicle but the patient dresses and undresses in the presence of the physician. As soon as finances permit a separate examining room should be provided.

The Waiting Room—The waiting room must naturally lead out into the entrance hall. In the home office during office hours the front parlor functions for professional purposes as the waiting room. In the evening the waiting room reverts to type as the sitting room for the family.

The waiting room is furnished according to the taste of the individual. Simplicity should be the keynote. Provision should be made for storming outside clothing particularly in inclement weather.

THE SEGREGATION OF PATIENTS—For diverse reasons it is sometimes essential to segregate patients, since certain of them do not wish to be seen by the other occupants of the waiting room. Pregnant women sometimes desire to conceal their condition. Patients with venereal diseases find difficulty explaining frequent visits to the doctor where families feud and marital partners become separated. Divorced and remarried factions ought not to meet in the common waiting room.

Many adults entertain a real phobia about communicable disease. Some patients not without justice regard the waiting room as a place where they will very likely be exposed to infectious and contagious diseases. The doting mother suspects that the child of every other mother suffers from whooping cough or tuberculosis.

It is distressing for the normally sensitive person to share the waiting room with unfortunates who have ugly wounds, paroxysmal cough or discharging sinuses, particularly if the lesions give off a putrid odor. The great unwashed may foul the air especially if the weather is inclement and the windows must be closed. The practitioner can avoid the criticism of the offended and the illwill of the unfortunates by giving special appointments to those whose presence in the waiting room might prove dangerous or offensive to fellow patients.

Despite all efforts awkward situations inevitably crop up. The most seasoned diplomat would find it difficult to placate all of the waiting patients, particularly those whose medical conditions are accompanied by anxiety. To these each waiting moment augments the sense of insecurity and proportionately increases their impatience.

The practitioner should indulge himself in the luxury of a secretary as soon as finances permit. An appointment system is introduced in the interest of the patients and for the well being of the physician. Despite this certain patients demand that they be seen immediately even if an emergency has to be manufactured. Nothing will prevent the paranoid and sensitive patient from feeling that he has been discriminated against and neglected.

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Franklin " Point Counter Point ' by Aldous Huxley Of Human Bondage by Somerset Maugham The Summing Up by Somerset Maugham The Life of Sir William Osler by Harvey Cushing Acquaintance and Other Essays by Sir William Osler 'Jürgen' by James Branch Cabell Lord Jim' by Joseph Conrad Rats Lice and History by Hans Zinnser 'Schweik the Good Soldier' by Hasek Candide by Voltaire The Home Book of Verse by B E Stevenson Abraham Lincoln by Carl Sandburg, 'Abraham Lincoln' by John Drinkwater Medical Essays

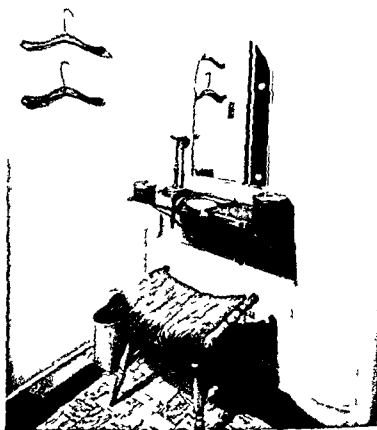


Fig. 1180—A simple practical and inexpensive method of converting a closet into a dressing room

and especially the essay on Puerperal Fever by Oliver Wendell Holmes Arrowsmith by Sinclair Lewis A Generation of Vipers' by Philip Wylie Mea Culpa by Celine especially the essay on Semmelweis and The Collected Poems of Walt Whitman

The Dressing and Examining Rooms—In the home the examining room either is part of the consulting room or it is placed in the former pantry or kitchen. This arrangement provides no circulation of and the doctor must wait for his patient to dress and

Examination

table is placed in a corner of the office the entire awkward performance takes place without the slightest pretense at privacy—much less with regard to modesty. A cupboard in the examining room is easily transformed into a satisfactory *dressing room* for electro diagnostic investigations (p. 3288).

Sooner or later the successful physician finds that he cannot countenance this archaic system of practice. His well being demand a separate office with at least two or three small examining or treatment rooms each



Fig. 1181.—A practitioner's examining room. Note the adjustable floor examining table (with footstool for patient, which cannot be seen in these pictures) revolving stool for the examiner weight and height scale wall sphygmomanometer wash basin. Another view appears in Fig. 1182.

of which is provided preferably with a cubicle or closet serving as a dressing room. With such an establishment several patients may be handled simultaneously and while one patient is dressing undressing or resting another may be seen examined or given treatment.

DRESSING ROOM.—The dressing room may be inexpensively and satisfactorily equipped with a glass shelf mirror and a small stool (Fig. 1180). The shelf is affixed to the wall at table height. The stool is placed in front of

the shelf and the mirror is hung directly above it. On the shelf are set out inexpensive appurtenances containing unscented talcum hairpins and safety pins a comb a hand mirror a shoe horn and button hook. The side wall at standing height is provided with clothes hooks and hangers.

If it is at all possible the dressing room should also have a toilet and a hand basin to serve the convenience of the patient and permit the collection of urine and stool specimens.



Fig 1180.—Another view of examining room in Fig 1181. Note electrical sterilizer and sterilizer table with provision for bottles on top and for storage of smaller material beneath overhead indirect lighting movable instrument table portable spot lamp cabinets for materials and supplies. There are also a refuse can with foot pedal and a wall bracket with varying widths of adhesive plaster which do not appear in these pictures.

EXAMINING ROOM (Figs 1181-2)—Particularly in the formal office separate rooms are available for the examination of patients. The illumination of the examining room is of great importance since it is rarely possible to use daylight. An overhead source of indirect lighting may be used for all general purposes a portable spot lamp is indispensable for the close examination of local lesions and the performance of technical procedures such as surgery.

The floor of the examining room should be covered with a hard surface which can be readily cleansed with a wet mop. Rugs and carpeting become stained and smelly and are to be avoided.

TABLE 954—FURNISHINGS AND EQUIPMENT OF THE EXAMINING ROOM

Hot Basin

Equipped with taps for hot and cold running water. On the adjacent wall a towel rack with clean towels or disposable tissues, a soap dispenser (or a soap dish) a hand brush and an orange stick.

Electric Sterilizer (Fig. 1182)

With three speeds and an automatic cut off that operates when the water level has fallen, preferably with cabinet stand.

Physicians who desire to sterilize dry dressings and gloves may purchase a fireless cooker. These sell at a price considerably less than the surgical autoclave.

Cotton gauze and packings may be purchased in packages previously sterilized.

Examining Table (Fig. 1182)

Metal tables are more easily cleaned and less likely to become moldy. The table should have a leather pad covered with clean linen or paper sheets. It should be adjustable so that the patient can be examined in the sitting position, lying flat or in the gynecological manner with the feet in the stirrups.

Provision should be made for drainage from the table into a slop pail so that irrigations can be conveniently practiced.

Arm of Stool

Placed before the examining table for the convenience of the patient (Fig. 1181).

Arm and Leg Stool

For the examiner, particularly for use during gynecological therapy (Fig. 1181).

Instrument Cabinet

With a lower compartment containing drawers for the storage of materials and a cabinet with glass doors enclosing shelves on which are placed the various instruments.

Portable Instrument Table

Of the standard type. Particularly useful when the practitioner has minimal nursing assistance. All of the necessary materials may be set out on the portable stand (Fig. 1182).

Portable Lamp

Of the floor type with a goose neck and hemispherical shade. Provides essential illumination during surgical procedures. The bulb should be at least 100 watt (Fig. 1182).

Refuse Can

Operated by a foot pedal and lined with a paper bag for the easy disposal of dressings.

Scale

Of the balance type with a measuring rod for estimating height. On the adjacent wall may be conveniently placed a height weight and age table.

Sphygmomanometer

Of the wall type. A great convenience provided the physician can afford an extra portable machine for the emergency bag (p. 3750).

Office Laboratory

For special equipment see p. 3660.

THE SPECIFICATIONS FOR THE EXAMINING ROOM—The examining room must contain technical equipment necessary for competent medical practice. In the majority of instances the limitations of medical practice result from inadequate physical equipment. Most missed diagnoses might

have been successfully made if the physician had had the proper utensils. The treatment of the patient is equally dependent upon the availability of apparatus. Most therapeutic procedures can be executed only where the required materials are readily at hand.

The Office Personnel—The proper maintenance of the office requires more than merely professional service. The tasks include housekeeping, secretarial work, nursing service and bookkeeping.

Housekeeping Service—In the professional office cleanliness, neatness and tidiness are mandatory. This is true not only from the standpoint of the meticulous patient but also as an economy in maintaining equipment and avoiding replacement of materials. In the home the physician's wife will commonly take care of the housekeeping. Many professional offices provide housekeeping service.

Secretarial and Nursing Service—In the home the wife or the house servant often doubles as the physician's helper. As soon as the budget will permit a neat, personable young woman should be employed as the 'aide' on a part time basis if necessary.

It is not essential that the aide be a highly trained secretary or a registered nurse. Her duties include the care of the waiting room, the arrangement of appointments, handling records, answering the telephone, the purchase and storing of equipment, interviewing the salesmen and other non professional visitors, assisting patients to dress and undress, passing instruments for minor procedures and straightening out the examining room after each patient has been dismissed.

As the office becomes busier the physician will need the services of a trained secretary and a registered nurse.

The *secretary* should be proficient in typing and stenography. Her duties include the transcription of the medical records, the care of the files for correspondence and the maintenance of the business records, particularly with a view to making out income tax returns.

The *nurse* assists the physician with his technical procedures. While the physician is otherwise occupied she may give injections, irrigations and carry out other similar procedures. She may with or without the physician go to the home of the patient and administer nursing care when patients cannot come to the office. The fees accruing from such services often cover her salary.

The practitioner who has the good fortune to be provided with competent secretarial and nursing assistance is freed of much of the cumbersome and time consuming routine of practice. He is enabled to occupy himself to much greater advantage with more important functions.

The Record System—On the very first day the physician opens his office he must solemnly resolve that he will keep accurate and complete records. It is a comparatively simple matter to inaugurate such a system at the beginning of practice. It is almost impossible to introduce systematic record keeping once practice has been started.

History Forms for Institutional Use—Routine history forms are commonly employed in medical schools and hospitals. They are necessary both for completeness and for filing purposes. These forms are usually printed with spaces for the subdivisions of the history or they list conditions to be checked, crossed or marked with the noxious word 'no'.

In private practice these forms prove to be a burden rather than a help the spacing is usually excessive or insufficient the habit of making checks crosses and negatives encourages mental laziness and makes of the historian a routinist rather than an intelligent auditor

Record Forms and Files in Private Practice—In private practice the record system must cover more than the medical history of the individual patient It embraces (1) the information necessary for the *business conduct* of the office (2) data for the computation of the various types of *income tax* (3) and accurate *reference for medicolegal matters* pertaining to court procedure insurance papers and claims of one type or another

THE MEDICAL RECORD—For the medical record the physician should use either a *stiff card* (no smaller than 5x8 and preferably 8½ x 11) enclosed in an envelope or a *leaflet* of four sides typewriter size (8½ x 11) enclosed in a stiff cardboard or manila folder

At the top of the first page of the record the *name of the physician* should be printed Beneath there should appear headings for the *name age marital state occupation address and telephone number of the patient* Extra sheets may be added when necessary

The envelope or folder should store letters correspondence reports and any other miscellany that refer to the individual patient For ease of filing the envelope or folder should have a tab for the name If filing is done alphabetically no other index is necessary except for special purposes such as diagnosis and therapy Steel files are to be preferred As soon as possible effort should be made to preserve all records in the typewritten form

The original history physical examination diagnosis and recommendations should be entered for the record Follow up notes particularly pertaining to house visits must be scrupulously recorded This is especially true of prescriptions and data for compensation and medicolegal purposes There is nothing more embarrassing than to have the patient request a prescription that his physician has forgotten or to learn that the prescription supposedly repeated has proven to be something wholly different such as capsules instead of powder or syrup of cherry instead of lemon

Meticulous care in record keeping richly pays the effort Nothing will so fix the loyalty of even an errant patient than the knowledge that in his physician's files are contained not only all of the medical records blood examinations and similar data but also copies of medications and prescriptions of worth

THE BUSINESS CARD—The business card may be either 4x6 or 5x8 It is separately filed in a drawer of the steel cabinet At the top appear the patient's name address and telephone number just as on the medical record Convenient charge cards may be purchased which contain vertical rulings for the days of the month and horizontal rulings for the months of the year providing a box for each day in the year Herein may be entered the charge for that day or a code letter indicating the service performed

Cards for patients whose bills are current may be filed separately in one drawer From these cards may be calculated the bills to be rendered When the indebtedness is discharged or the medical condition becomes inactive the cards may be restored to an inactive drawer These cards also serve for the computation of the various types of income tax

It is a wise precaution to reserve an adequate amount (at least 20 per cent) of all income in a special fund as a reserve for *tax payments*

LEDGER BOOK FOR RECORDING LABORATORY EXAMINATIONS AND TREATMENTS—A ledger book should be maintained for recording laboratory examinations and treatments. As soon as a service is rendered the name of the patient is entered, the date and the nature of the service recorded. When the examination or treatment has been completed, a report is entered later to be copied on the medical record. At this time the charge is posted on the business card.

LEDGER BOOK FOR BUSINESS PURPOSES—A second ledger book for purely business purposes contains the daily, weekly, monthly or yearly income. This records cash received, charges entered, bills sent, bills paid, bills outstanding and bad debts. In addition, place is reserved for notations of those expenses incurred that are legitimately deductible from the gross receipts in the calculation of the *income tax*.

INCOME TAX DEDUCTIONS—Income tax deductions include

- 1 The *rent*—either partially if at home or in full if in a professional building
- 2 The bills for *lighting and heating* the office
- 3 The *salaries* of the secretary, nurse, technician or maid
- 4 The *telephone bill*
- 5 The expenses incidental to *transportation* such as railroad, bus and taxi fares
- 6 The *upkeep of an automobile* (for professional use) including the cost of garaging, the purchase of gasoline and oil, repairs and service charges, license plates and insurance
- 7 *Medical dues and subscriptions*
- 8 *Expenses for traveling* to consultations, meetings, post graduate courses, etc.
- 9 The *fees expended for post graduate work*
- 10 The cost of *books and subscriptions*
- 11 Moneys paid to *doctors, physiotherapists or technicians*
- 12 *Certain insurance premiums* such as automobile and malpractice
- 13 Expenses (including narcotic and alcohol taxes) for *biologicals, drugs and chemicals* employed in the performance of tests or the treatment of the patient
- 14 *Capital expenses* such as the purchase of an automobile or physical equipment (X ray machine, diathermy apparatus, incubator or a microscope) may be depreciated according to the life expectancy of the product. The annual depreciation is deductible until the entire purchase price has been written off or until its resale. When resale occurs, as in the instance of an automobile, the total depreciation plus the resale price or allowance, must equal the purchase price.
- 15 Sales personal property, real estate, state or city (but not federal) income tax, gasoline tax, unemployment tax if assessed against physician as an employer
- 16 Losses due to accidents not covered by insurance

STATIONERY—Letter heads and envelopes prescription pads and professional cards may be chosen from the supply of the local printer The prescription pad in addition to name address telephone number and office hours should contain the *narcotic registry number*

BLANKS AND FORMS—The law requires that each physician secure a state license for practice and pay a *Federal narcotic and alcohol tax* The Federal government provides forms to be filled out when the physician orders or uses narcotic or alcohol In addition *death certificates birth certificates* cards for notification of *infectious and venereal diseases* forms for the *sending of specimens* (such as blood) to the local or state health departments are provided on request by the local health officers or their representatives Forms for *compensation cases* are also to be kept supplied

CALENDAR—A calendar of the visible type such as the Phillips Brooks should hang in a conspicuous place These calendars show the days of each month on a single page On them may be copied from year to year important dates that are apt to be forgotten Such for example might be dates for the payments of the income tax and insurance policies personal reminders for birthdays and anniversaries which the busy physician is apt to forget meeting nights are recorded and in addition the calendar is used as a reminder in the follow up of patients

VISIBLE FILE—A visible file should be kept with the name telephone number and address of patients nurses technicians physicians consultants the representatives of the supply companies and drug houses

It is a wise precaution to reserve an adequate amount (at least 20 per cent) of all income in a special fund as a reserve for tax payments

LEDGER BOOK FOR RECORDING LABORATORY EXAMINATIONS AND TREATMENTS—A ledger book should be maintained for recording laboratory examinations and treatments. As soon as a service is rendered the name of the patient is entered, the date and the nature of the service recorded. When the examination or treatment has been completed a report is entered later to be copied on the medical record. At this time the charge is posted on the business card.

LEDGER BOOK FOR BUSINESS PURPOSES—A second ledger book for purely business purposes contains the daily, weekly, monthly or yearly income. This records cash received, charges entered, bills sent, bills paid, bills outstanding and bad debts. In addition, place is reserved for notations of the expenses incurred that are legitimately deductible from the gross receipts in the calculation of the income tax.

INCOME TAX DEDUCTIONS—Income tax deductions include

- 1 The *rent*—either partially if at home or in full if in a professional building
- 2 The bills for *lighting and heating* the office
- 3 The *salaries* of the secretary, nurse, technician or maid
- 4 The *telephone bill*
- 5 The expenses incidental to *transportation* such as railroad, bus and taxi fares
- 6 The *upkeep of an automobile* (for professional use) including the cost of garaging, the purchase of gasoline and oil, repairs and service charges, license plates and insurance
- 7 *Medical dues and subscriptions*
- 8 *Expenses for traveling* to consultations, meetings, post graduate courses, etc.
- 9 *The fees expended for post graduate work*
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- Cabot rings** Circular or figure-of-eight nuclear remnants in immature erythrocytes
- Capillary fragility tests** In Hemorrhagic diatheses
- Chauffard Minkowski's disease** Chronic familial hemolytic jaundice
- Chloroma** Greenish tumorlike infiltrations in the bones of the skull in children with acute leukemia
- Chlorosis.** Iron deficiency anemia in adolescent girls
- Cold hemagglutination** Agglutination of red blood cells by serum only in the cold
- Color index.** With 60 per cent H_z and 4 000 000 cells the index is

$$\frac{60}{40 \times 2} = \frac{60}{80} = 0.75$$

- Congestive splenomegaly** A clinical syndrome with splenomegaly anemia leukopenia thrombocytopenia and gastric hemorrhages
- Cooke count** Simplification of the Arneith count
- Cooley's anemia** Fatal erythroblastic anemia of young children (leuko-erythroblastic anemia of childhood Mediterranean anemia Thalassaemia)
- Crenation** Shrivelling of red cell because of hypertonicity of surrounding medium
- Degenerative index.** Ratio of degenerated leukocytes *i.e.*, those showing toxic granulation or vacuolization
- Donath-Landsteiner reaction** Hemolysis resulting from the warming of chilled blood in cold paroxysmal hemoglobinuria
- Donovan bodies.** In Leishmaniasis
- Dorothy Reed cells.** Also called *Sternberg cells* Multinucleated cells in Hodgkin's disease
- Drepanocytic anemia** See *Sickle cell anemia*

- Egyptian splenomegaly** Use *Schistosomiasis splenomegaly*
- Elliptocytes.** Oval red cells Use *Ovalocytes*
- Eosinophilia** Increased number of eosinophilic leukocytes
- Eosinophilic.** Takes eosin (red) stain as in normal erythrocytes
- Erythremia** Use *Polycythemia vera*
- Erythroblast** Immature erythroid cell of the bone marrow
- Erythroblastic anemia of Cooley** Mediterranean anemia
- Erythroblatous.** Increase in erythroblasts in the bone marrow
- Erythroblastous foetalis.** Hemolytic anemia of the neonatal period usually due to the Ph factor Often associated with icterus gravis and universal edema
- Erythrocyte maturation factor** Principle causing maturation of erythrocytes
- Erythrocytosis.** Increase in red blood cell count, usually a result of dehydration See *Secondary polycythemia*
- Erythroid** Referring or pertaining to the red blood cell producing tissue
- Erythro-leukemia polycythemia** Terminating as leukemia
- Erythron.** All red cells and tissues which form them
- Erythrophagocytosis.** Phagocytosis of red cells by leukocytes in some acute hemolytic anemias

GLOSSARY OF HEMATOLOGY

- Acholic jaundice** Jaundice without bile in the urine as in hemolytic jaundice
- Achrestic anemia** Fatal macrocytic hyperchromic anemia
- Achromia** A misnomer which usually refers to a diminution in the hemoglobin of the erythrocytes Use *Hypochromia*
- Achylia** Almost complete absence of gastric juice as seen in macrocytic hyperchromic anemia
- Addisonian anemia** Use *Macrocytic hyperchromic anemia* p 1077
- Agglutinin** An antibody which combines with an agglutino-gen to cause agglutination
- Agglutino-gen** A receptor substance in the red blood cells with which an agglutinin combines
- Agranulocytic angina** See *Agranulocytosis*
- Agranulocytosis** A clinical syndrome with marked reduction in total leukocytes of the blood and almost complete disappearance of the granulocytes
- Aleukemia** Leukemia with a normal or diminished white blood count
- Anemia** A reduction in the hemoglobin content of the blood
- Anisocytosis** Variation in size of the erythrocytes
- Anti anemic principle** Use *Hematopoietic principle* or *Erythrocyte maturation factor*
- Aplasia** Incomplete or defective development cessation of regeneration
- Aplastic ananemia** See *Marrow anemia* p 1090
- Aregenerative anemia** See *Marrow anemia*
- Arneth count** Count of the polymorphonuclear leukocytes according to the configuration and number of nuclear lobes
- Auer bodies** Metachromic rodlike granules in myeloblasts
- Autoagglutination** Use *Cold hemagglutination* Agglutination of erythrocytes by their own serum
- Ayerza's syndrome** Secondary polycythemia due to stenosis of pulmonary arteries ("black cardiacs")
- Band form** Juvenile stab or staff polymorphonuclear leukocyte with an indented but nonsegmented nucleus
- Banti's syndrome** Use *Congestive splenomegaly*
- Basophilia** Immature red cells which take blue stain
- Basophilic** Takes the basic or blue stain
- Bence-Jones proteosuria or proteinuria** Heat labile proteinuria in multiple myeloma
- Biermer's anemia** Use *Macrocytic hyperchromic anemia* p 1077
- Bilirubin** Bile pigment
- Blackwater fever** Estivo autumnal malaria with hemoglobinuria
- Blood crisis or blast crisis** Sudden appearance of large numbers of nucleated erythrocytes in peripheral blood in macrocytic and hemolytic anemias

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- Fabism (favism)** Acute hemolytic anemia in Italy due to hypersensitivity to fava bean
- Familial hemolytic jaundice** Also **Familial spherocytosis** and **Chaufford Minkowski disease** Increased fragility of red cells spherocytosis and splenomegaly
- Familial nonhemolytic jaundice** Hereditary mild dysfunction of liver with out anemia
- Felty syndrome** See *Granulocytopenias*
- Fibrinogen** Soluble plasma protein changed to fibrin by thrombin
- Filament count** The percentage of polymorphonuclear leukocytes with segmented nuclei
- Follicular lymphoblastoma** Disease of the lymphatic tissue characterized by giant lymphoid follicles
- Gaucher's disease** Metabolic lipid reticulo endotheliosis with kersin deposition in the organs
- Geisböck's disease** A primary polycythemia vera with hypertension but without splenomegaly
- Geographical skull** Roentgen appearance in Hand Christian Schuller syndrome
- Glandular fever** Use *Infectious mononucleosis* Synonymous with *benign lymphadenosis*
- Goat's milk anemia** See *Iron deficiency anemia*
- Granulocytes** Granular leukocytes of neutrophilic basophilic and eosinophilic varieties
- Granulocytopenia** Used synonymously with agranulocytosis agranulocytic angina malignant neutropenia
- Granulocytosis** See *Leukocytosis*
- Haff disease** See *Myoglobinuria* p 1076
- Ham test** The hemolysis of red cells by fresh acidified human serum pathognomonic of nocturnal paroxysmal hemoglobinuria
- Hand Christian Schüller syndrome** A xanthomatosis with defects in skull bones exophthalmos and diabetes insipidus
- Hayem Widal disease** Acquired hemolytic anemia
- Hemacytometer** Instrument for counting red cells
- Hemagglutination** Agglutination of red blood cells
- Hematocrit** Volume of packed red blood cells
- Hematogone** An immature cell of the bone marrow, whose exact function is not known Present in large numbers in follicular lymphoblastoma
- Hematopoietic principle** Substance in liver which accomplishes maturation of erythrocytes
- Hemoconcentration** Decrease in blood plasma in instances of marked fluid loss
- Hemoconia** Blood dust
- Hemocytoblast** Most immature cell of the bone marrow giving rise to all the bone marrow elements
- Hemoglobinemia** Hemoglobin pigment free in blood plasma
- Hemoglobinuria** Hemoglobin pigment free in the urine (Port wine urine or Blackwater")

- Hemogram Complete blood count including differential blood count
- Hemolysis Destruction of red blood cells
- Hemophilia See *Hemorrhagic diatheses* p 1108
- Hemorrhagic capillary toxicosis See *Capillary disturbances* p 1121
- Hemorrhagic disease of the newborn Vitamin K deficiency
- Hemorrhagic telangiectasia (Rendu Osler Webers disease) Congenital malformation of the capillaries with a tendency to bleed
- Hemosiderin Iron containing fraction of hemoglobin which gives the Prussian blue reaction
- Hemosiderinuria Present only in hemochromatosis and Marchiafava Micheli syndrome
- Henoch Schonlein purpura See *Hemorrhagic capillary toxicosis* p 1121
- Heparin Anti prothrombin in blood and organs possibly derived from the mast cells
- Heterophile antibodies Appear in blood in infectious mononucleosis and cause agglutination of sheep erythrocytes
- Hirudin Antithrombin obtained from leeches
- Histoplasmosis Infectious fungus disease with fever splenomegaly and generalized lymphadenopathy Parasites present in cells of peripheral blood
- Hodgkin's disease See *Diseases of lymph nodes*
- Howell Jolly bodies Nuclear remnants in young erythrocytes
- Hunterian glossitis See *Macrocytic hyperchromic anemia*
- Hydrops foetalis See *Erythroblastosis foetalis*
- Hyperleukocytosis Marked increase in white blood cells seen in very severe infections or acute hemolytic anemias
- Hyperchromic Having an increased hemoglobin concentration (usually used with macrocytes with normal concentration of hemoglobin)
- Hyperplasia Increased activity
- Hypochromic Having a decreased hemoglobin concentration
- Hypoplasia Decreased activity
- Hypoprothrombinemia See *Hemorrhagic diseases*
- Icterus gravis Use *Erythroblastosis foetalis*
- Infectious mononucleosis See *Generalized infections*
- Intrinsic factor Substance in gastric juice necessary for normal blood development
- Isoagglutination Agglutination of the red blood cells according to the blood group scheme
- Juvenile neutrophil Nonsegmented polymorphonuclear leukocyte metamyelocyte
- Kahler's disease Use *Multiple myeloma* or *Myelomatosis*
- Kernicterus Icteric staining of the basal ganglia especially in erythroblastosis foetalis
- Koilonychia Softening and deformity of the fingernails as seen in idiopathic hypochromic anemia

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- Hemoglobinuria** Hemoglobin pigment free in the urine (Port wine urine or Blackwater')

- Myelogenous** See *Myeloid*
- Myeloid** Referring or pertaining to tissue producing white blood cells (usually granular leukocytes)
- Myeloma** Malignant hematopoietic disease usually due to an increase in plasma cells and rarely other cells
- Myelophthisic anemia** See *Marrow anemias*
- Myelosarcoma** Myeloma with markedly invasive and destructive qualities
- Myoglobinuria** See *Hemoglobinurias*
- Neutropenia** See *Agranulocytosis*
- Niemann Pick's disease** Reticulo-endotheliosis of infancy with phosphatide deposition in the organs
- Nocturnal paroxysmal hemoglobinuria** Marchiafava Micheli syndrome
- Normoblast** Nucleated erythroid cell of the bone marrow between the erythroblast and erythrocyte in development
- Normoblastemia** Normoblasts in the circulating blood
- Normoblastosis** Increase in normoblasts in the bone marrow
- Normochromic** Having normal hemoglobin concentration
- Normocyte** Normal size erythrocyte
- Oroya fever** Caused by *Bartonella bacilliformis* and characterized by a severe macrocytic hemolytic anemia (Carrion's disease)
- Osteosclerosis** Increase in density of the bones may be associated with anemia
- Ovalocyte** Oval shaped red blood cell
- P A factor** Use *Hematopoietic principle*
- Panmyelosis** Proliferation of all bone marrow cells as seen in chronic myeloid leukemia
- Paroxysmal hemoglobinuria** Attacks in which port wine urine is voided
- Paul Bunnell test** Test for heterophile antibodies
- Pelger Huet phenomenon** Constitutional abnormality of the leukocytes with a shift to the left in the differential blood count
- Pernicious anemia** Use *Macrocytic hyperchromic anemia*
- Peroxidase stain** Demonstrates granules in immature (but not in myeloblasts) myeloid cells and monocytes
- Plasma cell** Present in chronic infections and in multiple myeloma
- Plummer Vinson syndrome** Glossitis dysphagia and anemia in idiopathic hypochromic anemia
- Phlebothrombosis** See *Intravascular clotting*
- Plasma volume** Total amount of blood plasma in the body
- Poikilocytosis** Variation in shape of erythrocytes
- Polychromatophilia** Bluish color to erythroid cells sign of immaturity (polychromasia) equivalent to reticulocytosis
- Polycythemia [polyglobulia]** Clinical syndrome characterized by marked increase in the red blood cell mass
- Polyphyletism** Hematologic theory that assumes there are several stem cells
- Price-Jones curve** Graph showing the measured diameters of the red blood cells

- Lederer's anemia** Acute hemolytic anemia of undetermined cause in children
- Letterer-Siwe's disease** A reticulo endotheliosis of infancy
- Leukemia** Malignant tumor of the hematopoietic system involving the leukocytes
- Leukocyte** White blood cell
- Leukocythemia** Marked increase in white blood cells as in leukemia
- Leukocytosis** Increase in white blood cells above normal
- Leukopenia** Decrease in white blood cells below normal
- Leukosarcoma** Leukemia with markedly invasive qualities
- Lipoid histiocytosis** See *Niemann Pick's disease*
- Loeffler's syndrome** Transient rapidly shifting pulmonary infiltrations with few symptoms but with an eosinophilia of 15 to 45 per cent
- Lymphadenopathy** Generalized enlargement of lymph nodes
- Lymphocytosis** Increase of lymph cells in peripheral blood
- Lymphosarcoma** Malignant tumor of lymphatic origin
- Macrocyte** Erythrocyte larger than normal
- Macropolycyte** Large markedly segmented polymorphonuclear leukocyte of macrocytic hyperchromic anemia
- Marchiafava-Micheli syndrome** Chronic hemolytic anemia with hemosiderinuria and nocturnal hemoglobinuria
- Maturation arrest** Cessation of growth of bone marrow cells at an immature level because of the absence of a maturation factor
- Maturation factor** A substance which will cause cells to ripen to maturity
- Mean corpuscular volume** Volume of the average red blood cell
- Mediterranean anemia** Use *Cooley's anemia*
- Megakaryoblast** Precursor of the megakaryocyte
- Megakaryocyte** Giant bone marrow cells from which blood platelets arise
- Megakaryophthisis** Decrease and anatomical abnormalities in the megakaryocytes
- Megaloblast** Early bone marrow cell of erythroid series found only in the fetus or in liver extract deficiency anemias
- Megalocyte** Use *Macrocyte*
- Meniscocytosis** Sicklemia or if anemia sickle cell anemia
- Metamyelocyte** Neutrophilic basophilic and eosinophilic maturing cell of the myeloid series between myelocyte and nonsegmented polymorphonuclear leukocyte
- Methemalbumin** Oxidized heme combined with blood serum albumen
- Methemoglobin** Oxidized hemoglobin in which the iron is in the trivalent state
- Microcyte** Erythrocyte smaller than normal
- Mononucleosis** Usually infectious mononucleosis
- Monophyletism** Theory that all blood cells have common origin
- Morbus maculosus werlhofi** See *Essential thrombocytopenic purpura*
- Myeloblast** Immature cell of the myeloid series from which the granular leukocytes arise
- Myelocyte** Neutrophilic basophilic and eosinophilic immature cell of the myeloid series between promyelocyte and metamyelocyte
- Myelofibrosis** Fibrosis of the bone marrow

- Thrombocyte Blood platelet
- Thrombocythemia Use *Thrombocytosis*
- Thrombocytopenia Reduction in blood platelets below normal
- Thrombocytopenic purpura Hemorrhagic disease benefited by splenectomy
- Thrombocytosis Increase in blood platelets above normal
- Thrombokinas Use *Thromboplastin*
- Thromboplastin Substance liberated by disintegration of platelets or by extravascular tissues which initiates blood coagulation
- Toxic granulation Dark blue coarse granulation of polymorphonuclear leukocytes in toxic conditions
- Turk's cell Variation of plasma cells or atypical lymphocyte
- Universal donor Blood donor of blood group O whose cells are nonagglutinable
- Universal edema Edema associated with erythroblastosis foetalis
- Universal recipient Patient of blood group AB whose serum is free of isoagglutins
- Urobilinogen Iron free excretion end product of hemoglobin metabolism Increased in urine and stool in hemolytic anemia Increased in urine only in diffuse liver disease
- Vaquez Osler disease Use *Polycythemia vera*
- Vitamin C—cevitamic acid See *Scurvy*
- Vitamin K—naphthoquinone See *Hypoprothrombinemia*
- Vitamin P Citrin
- Von Jaksch's anemia Childhood anemia with leukerythroblastosis
- Werlhof's disease Primary thrombocytopenic purpura
- Widal's hemoclasia crisis Leukopenia after protein meal
- Winckel's disease Rare hemolytic syndrome of the newborn characterized by cyanosis jaundice and hemoglobinemia
- Xanthomatosis See *Icticulo endotheliosis*

- Primary anemia** Macrocytic hyperchromic anemia
- Prothrombin** Present in nonclotted blood and forms thrombin in presence of thromboplastin and ionic calcium
- Pseudoagglutination** Use *Rouleau*
- Purpura** Hemorrhage into the tissues
- Purpura hemorrhagica** Use *Thrombocytopenic purpura* may be primary or secondary
- Pyknosis** Condensation of nuclear chromatin into a dense mass before extrusion
- Red blood cell mass** Total volume of red blood cells in the body
- Rendu Osler Weber disease** Use *Hereditary hemorrhagic telangiectasis*
- Reticulocyte** Immature erythrocyte containing a stainable reticulum
- Reticulo endothelial system** Widespread group of cells and fibers with unusual phagocytic properties in all of the organs of the body but mainly in the bone marrow, liver lymph nodes and spleen
- Reticulo endotheliosis** Proliferation of the cells of the reticulo endothelium
- Rh factor** Antigen present in 85% normal human red blood cells Named for *rhesus* monkeys
- Rouleau formation** Formations of red blood cells like coins
- Rumpel Leede test** See *Capillary fragility tests*
- Scurvy** Vitamin C deficiency
- Secondary anemia** Anemia of known cause State cause but do not use term
- Shift to the left** Increase in immature glandular cells (nonsegmented polymorphonuclear leukocytes)
- Shift to the right** Increase in mature segmented polymorphonuclear cells
- Schilling hemogram** Differential blood count separating juvenile stab and segmented polymorphonuclear cells
- Sickle cell** Abnormal erythrocyte which is thinned and resistant to hemolysis
- Smear or smudge cells** Degenerated lymphocytes common in chronic lymphatic leukemia
- Spherocyte** Red blood cell in which the ratio of thickness to diameter is increased markedly fragile to hypotonic saline
- Spherocytosis** See *Familial hemolytic jaundice*
- Splenic anemia** See *Congestive splenomegaly*
- Splenic granulocytopenia** See *Granulocytopenias*
- Stem cell** Use Hemocytoblast
- Supravital stain** Stains living cells of restricted value to differentiate and study blood cells
- Target cells (leptocytes)** Thin erythrocytes with marked resistance to hemolysis in hypotonic saline found in Cooley's anemia sickle cell anemia target cell anemia and other conditions
- Thalassemia** See *Erythroblastic anemia of Cooley*
- Thrombasthenia** Blood platelets with feeble coagulative function
- Thrombin** Enzyme formed from an interaction of prothrombin ionic calcium and thromboplastin which changes fibrinogen to fibrin

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—*Continued*

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|--------------------------------|---|---|
| Amalgam makers | Mercury and its compounds | History mercury in urine |
| Amber workers | Lead and its compounds | History lead in urine |
| Ammonia workers | Ammonia calcium cyanamide (cyanamide) carbon monoxide | History |
| Ammonium salt makers | Heat ammonia carbon disulfide cyanogen compounds hydrochloric acid sulfuric acid | History |
| Ammonium sulphate makers | Sulfuric acid | History |
| Amyl acetate workers | Amyl acetate amyl alcohol | History |
| Amyl alcohol workers | Amyl alcohol | History |
| Amyl nitrite workers | Amyl alcohol | History |
| Aniline dye makers | See Dye makers | History |
| Aniline workers | Aniline and other amino compounds of benzol and its homologues arsenic retted hydrogen (arsine) benzol (benzene) and its homologues (toluol and xylol) chromium compounds hydrochloric acid nitrobenzol and other nitro compounds of benzol and its homologues nitrous fumes and nitric acid | History arsenic in urine |
| Animal handlers | Anthrax fungous infectious septic infections | History |
| Annealers | Heat ammonia | History |
| Antifreeze makers | Methanol (methyl alcohol) | History |
| Antimony extractors (refiners) | Heat antimony and its compounds | History |
| Antimony fluoride extractors | Hydrofluoric acid | History |
| Antipyrine makers | Phenylhydrazine | History hemogram |
| Arsenic roasters | Heat arsenic and its compounds (except arsenuretted hydrogen) | History arsenic in urine |
| Art-glass workers | Amyl acetate benzine (naphtha gasoline) hydrofluoric acid lead and its compounds methanol (methyl alcohol) turpentine | History lead in urine |
| Artificial amber makers | Formaldehyde | History |
| Artificial flower makers | Repeated motion, pressure shock, etc arsenic and its compounds (except arsenuretted hydrogen) chromium compounds lead and its compounds mercury and its compounds methanol (methyl alcohol) | History arsenic in urine lead and mercury in urine hemogram |
| Artificial gem makers | Thallium | History |
| Artificial ice makers | Sudden variations in temperature dampness ammonia sulfur dioxide | History |
| Artificial leather workers | Heat acetone amyl acetate aniline and other amino compounds of benzol and its homologues arsenic and its compounds (except arsenuretted hydrogen) benzol (benzene) and its homologues (toluol and xylol) butanone (methyl ethyl ketone) butyl alcohol methanol (methyl alcohol) nitrous fumes and nitric acid sulfuric acid | History arsenic in urine hemogram |
| Artificial pearl makers | Acetone amyl acetate lead and its compounds nitrous fumes and nitric acid tetrachlorethane (acetylene tetrachlorid) | History lead in urine |
| Artificial stone makers | Tar and pitch | History |
| Asbestos miners | Asbestos See also <i>Miners</i> | History radiograph of chest, temperature record |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES*

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|--------------------------------|--|--|
| Abrasive workers | Heat organic dust inorganic dust inorganic dust containing no free silica | History radiograph of chest temperature record |
| Acetaldehyde workers | Acetaldehyde mercury and its compounds | History mercury in urine |
| Acetanilid workers | Aniline and other amino compounds of benzol and its homologues | History |
| Acetic acid makers | Hydrochloric acid mercury and its compounds | History mercury in urine |
| Acetone workers | Acetone mercury and its compounds | History |
| Acetylene workers | Inorganic dust (except asbestos) containing no free silica acetone ammonia arseniuretted hydrogen (arsine) carbon disulfide carbon monoxide chloride of lime chromium compounds See also <i>Carbide makers and welders</i> | History chest radiograph temperature record arsenic in urine |
| Acid dippers | Dampness arseniuretted hydrogen (arsine) cyanogen compounds hydrochloric acid nitrous fumes and nitric acid sulfuric acid | History arsenic in urine |
| Acid finishers (glass) | Hydrochloric acid lead and its compounds sulfuric acid | History lead in urine |
| Acridine workers | Acridine | } History |
| Acrolein workers | Acrolein | |
| Actors | Lead and its compounds | History lead in urine |
| Air hammer operators | Repeated motion pressure shock, etc | History |
| Airplane-dope makers | Acetone amyl acetate benzol (benzene) and its homologues (toluol and xylol) carbon tetrachloride formic acid (see also formaldehyde) methyl cellosolve (ethylene glycol monomethyl ether) tetrachlorethane (acetylene tetrachloride) | |
| Airplane-hangar projectors | Benzine (naphtha gasoline) benzol (benzene) and its homologues (toluol and xylol) carbon tetrachloride | History |
| Airplane pilots | Altitude rarefied air (decreased atmospheric pressure) carbon monoxide | History |
| Airplane pilots (crop dusting) | Altitude rarefied air (decreased atmospheric pressure) arsenic and its compounds (except arseniuretted hydrogen) carbon monoxide lead and its compounds | History arsenic in urine |
| Alcohol-distillery workers | Amyl acetate amyl alcohol benzol (benzene) and its homologues (toluol and xylol) mercury and its compounds methanol (methyl alcohol) | History mercury in urine |
| Aldehyde pumpmen | Acetaldehyde methanol (methyl alcohol) | History |
| Alkali salt makers | Dampness carbon dioxide chlorine hydrochloric acid sulfur dioxide sulfuretted hydrogen (hydrogen sulfide) | History |
| Alloy makers | Heat beryllium carbon monoxide cobalt magnesium manganese nickel selenium compounds vanadium | History |
| Aluminum extractors | Hydrofluoric acid manganese | History |
| Alum workers | Sulfuric acid | History |

* Compiled from Bulletin No. 41 U. S. Department of Labor

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|--|---|--|
| Bevelers | Inorganic dust (except asbestos) containing no free silica | History radiograph of chest temperature record |
| Billet mill workers (iron and steel) | Heat | History |
| Basque-kiln workers | Heat inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica carbon monoxide | History radiograph of chest temperature record |
| Blacksmiths | Heat ultraviolet and infra red rays repeated motion pressure shock etc carbon dioxide carbon monoxide cyanogen compounds lead and its compounds | History lead in urine |
| Blanket makers | Infection | History |
| Blasters | Inorganic dust containing free silica, inorganic dust (except asbestos) containing no free silica carbon monoxide nitrous fumes and nitric acid sulfuretted hydrogen (hydrogen sulfide) | History radiograph of chest temperature record |
| Blast furnace workers | Heat, carbon dioxide carbon monoxide cyanogen compounds phosphuretted hydrogen (phosphine) sulfur dioxide sulfuretted hydrogen (hydrogen sulfide) | History |
| Bleachers | Heat sudden variations in temperature chloride of lime chlorine chromium compounds hydrochloric acid hydrofluoric acid nitrous fumes and nitric acid oxalic acid ozone phosgene potassium hydroxide sodium hydroxide sulfur dioxide | History |
| Bleachery driers | Heat | History |
| Bleaching powder makers | Arsenuretted hydrogen (arsine) chloride of lime chlorine manganese | History arsenic in urine |
| Blockers (felt hats) | Heat carbon monoxide | History |
| Blooders (tannery) | Lead and its compounds | History lead in urine |
| Bloomers mill workers (iron and steel) | Heat | History |
| Blowers (felt hats) | Organic dust mercury and its compounds | History mercury in urine |
| Blowers-out (zinc smelting) | Heat zinc | History |
| Blue print makers | Chromium compounds | History |
| Blue print paper makers | Aniline and other amino compounds of benzol and its homologues oxalic acid | History |
| Blurs (revolvers) | Heat | History |
| Boiler cleaners and washers | Dampness carbon monoxide | History |
| Boiler room workers | Heat carbon dioxide carbon monoxide | History |
| Bonelack makers | Ammonia phosphorus | History |
| Bone renderers, extractors etc | Organic dust anthrax, acrolin cyanogen compounds sulfur dioxide | History |
| Bookbinders | Acrolein amyl acetate arsenic and its compounds (except arsenuretted hydrogen) lead and its compounds methanol (methyl alcohol) oxalic acid | History arsenic and lead in urine |
| Bottle-cap makers | Lead and its compounds | History lead in urine |
| Bottlers (mineral waters) | Carbon dioxide sulfuretted hydrogen (hydrogen sulfide) | History |
| Brake-lining makers | Asbestos benzol (benzene) and its homologues (toluol and xylol) | History radiograph of chest temperature record |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|---|---|---|
| Asbestos products workers | Heat asbestos benzol (benzene) and its homologues (toluol and xylol) for maldehyde tar and pitch | History radiograph of chest temperature record hemogram |
| Ashmen | Organic dust inorganic dust (except asbestos) containing no free silica | History radiograph of chest temperature record |
| Asphalt workers | Heat tar and pitch | History |
| Automobile painters | Dampness benzol (benzene) and its homologues (toluol and xylol) methanol (methyl alcohol) See also Painters | History hemogram |
| Automobile radiator cleaners | Oxalic acid | History |
| Aviation personnel (flying) | Altitude and rarefied air (decreased atmospheric pressure) See also Airplane pilots | History |
| Babbitt metal workers | Antimony and its compounds lead and its compounds | History antimony and lead in urine |
| Babbitters | Antimony and its compounds lead and its compounds | History antimony and lead in urine |
| Bacteriologists | Infections anthrax | History |
| Bakers | Sudden variations of temperature organic dust ultraviolet and infra red rays carbon dioxide carbon monoxide | History |
| Baking powder makers | Carbon dioxide | History |
| Balloon (hydrogen) workers | Arsenuretted hydrogen (arsine) | History arsenic in urine |
| Balloon inflators | Carbon monoxide | History |
| Barbers | Infection repeated motion pressure shock etc | History |
| Barium carbonate makers | Barium sulfuretted hydrogen (hydrogen sulfide) | History |
| Bar mill workers (iron and steel) | Heat | History |
| Barometer makers | Mercury and its compounds | History mercury in urine |
| Basic slag (artificial manure) workers | Inorganic dust (except asbestos) containing no free silica | History radiograph of chest temperature record |
| Batters (tannery) | Anthrax | History |
| Bathhouse attendants | Infection | History |
| Beaters (textiles) | Organic dust | History radiograph of chest temperature record |
| Beamhouse workers (tannery) | Dampness anthrax | History |
| Beatermen (paper and pulp) | Dampness chlorine | History |
| Beauty parlor operatives | Infection benzol (benzene) and its homologues (toluol and xylol) | History hemogram |
| Bed rubbers (stone) | Inorganic dust containing free silica in organic dust (except asbestos) containing no free silica | History radiograph of chest temperature record |
| Benzene workers | Benzene (naphtha gasoline) | History hemogram |
| Benzol purifiers | Benzol (benzene) and its homologues (toluol and xylol) sulfuric acid | History hemogram |
| Benzol stillmen | Heat benzol (benzene) and toluol and xylol | History |
| Beryllium alloy workers | Beryllium | History |
| Beryllium extractors | Hydrofluoric acid | History |
| Bessemer converter workers (iron and steel) | Heat carbon monoxide | History |
| Bets still operators (bets naphthol) | Heat sulfuric acid | History |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|--|--|---|
| Bevelers | Inorganic dust (except asbestos) containing no free silica | History radiograph of chest temperature record |
| Billet mill workers (iron and steel) | Heat | History |
| Busque-kiln workers | Heat inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica carbon monoxide | History radiograph of chest, temperature record |
| Blacksmiths | Heat ultraviolet and infra red rays repeated motion pressure shock etc carbon dioxide carbon monoxide cyanogen compounds lead and its compounds | History lead in urine |
| Blanket makers | Infection | History |
| Blasters | Inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica carbon monoxide nitrous fumes and nitric acid sulfuretted hydrogen (hydrogen sulfide) | History radiograph of chest temperature record |
| Blast furnace workers | Heat, carbon dioxide carbon monoxide cyanogen compounds phosphuretted hydrogen (phosphine) sulfur dioxide sulfuretted hydrogen (hydrogen sulfide) | History |
| Bleachers | Heat, sudden variations in temperature chloride of lime chlorine chromium compounds hydrochloric acid hydrofluoric acid nitrous fumes and nitric acid oxalic acid ozone phosgene potassium hydroxide sodium hydroxide sulfur dioxide | History |
| Bleachery driers | Heat | History |
| Bleaching powder makers | Arsenuretted hydrogen (arsine) chloride of lime chlorine manganese | History arsenic in urine |
| Blockers (felt hats) | Heat carbon monoxide | History |
| Blooders (tannery) | Lead and its compounds | History lead in urine |
| Blooming mill workers (iron and steel) | Heat | History |
| Blowers (felt hats) | Organic dust mercury and its compounds | History mercury in urine |
| Blowers-out (zinc smelting) | Heat zinc | History |
| Blue print makers | Chromium compounds | History |
| Blue print paper makers | Aniline and other amino compounds of benzol and its homologues oxalic acid | History |
| Bluers (revolvers) | Heat | History |
| Boiler cleaners and washers | Dampness carbon monoxide | History |
| Boiler room workers | Heat carbon dioxide carbon monoxide | History |
| Boneblack makers | <i>Ammonia phosphorus</i> | History |
| Bone renderers extractors etc | Organic dust anthrax acrolein cyanogen compounds sulfur dioxide | History |
| Bookbinders | Acrolein amyl acetate arsenic and its compounds (except arsenuretted hydrogen) lead and its compounds methanol (methyl alcohol) oxalic acid | History arsenic and lead in urine |
| Bottle-cap makers | Lead and its compounds | History lead in urine |
| Bottlers (mineral waters) | Carbon dioxide sulfuretted hydrogen (hydrogen sulfide) | History |
| Bottle-lining makers | Asbestos benzol (benzene) and its homologues (toluol and xylol) | History radiograph of chest, temperature record |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|-------------------------|---|---|
| Brass foundries | Heat antimony and its compounds arsenic and its compounds (except arseniuretted hydrogen) carbon dioxide carbon monoxide copper lead and its compounds phosphorus sulfur dioxide | History antimony arsenic and lead in urine |
| Brass polishers | Lead and its compounds See also <i>Polishers and cleaners (metal)</i> | History lead in urine |
| Braziers | Heat ultraviolet and infra red rays lead and its compounds zinc | History lead in urine |
| Brewers | Heat sudden variation in temperatures fungous infections amyl alcohol carbon dioxide carbon monoxide formaldehyde hydrofluoric acid phenol sulfuric acid | History |
| Brick burners | Heat carbon dioxide carbon monoxide lead and its compounds | History lead in urine |
| Bricklayers | Inorganic dust (except asbestos) containing no free silica | History radiograph of chest temperature record |
| Brick makers | Heat, dampness inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica, by hydrofluoric acid lead and its compounds magnesium manganese sulfur dioxide | History radiograph of chest temperature record |
| Briquet makers | Arsenic and its compounds (except arseniuretted hydrogen) tar and pitch | History arsenic in urine |
| Bromine makers | Aniline and other compounds of benzol and its homologues bromine chlorine | History bromine in urine |
| Bronze powder makers | Acetone zinc | History |
| Bronzers | Inorganic dust (except asbestos) containing no free silica ammonia amyl acetate arsenic and its compounds (except arseniuretted hydrogen) arseniuretted hydrogen (arsine) benzine (naphtha gasoline) benzol (benzene) and its homologues (toluol and xylol) cyanogen compounds, hydrochloric acid lead and its compounds manganese mercury and its compounds methanol (methyl alcohol) sulfuretted hydrogen (hydrogen sulfide) zinc | History arsenic in urine hemogram |
| Broom makers | Organic dust anthrax, chlorine formaldehyde sulfur dioxide | History |
| Browniers (gun barrels) | Cyanogen compounds lead and its compounds mercury and its compounds petroleum (see also <i>Benzine</i>) | History lead and mercury in urine |
| Brushers (felt hats) | Organic dust mercury and its compounds | History mercury in urine |
| Brush makers | Organic dust anthrax formaldehyde lead and its compounds methanol (methyl alcohol) tar and pitch | History lead in urine |
| Buffers | Defective illumination organic dust in organic dust containing free silica in organic dust (except asbestos) containing no free silica | History radiograph of chest, temperature record |
| Buffers (rubber) | Amyl acetate benzine (naphtha gasoline) lead and its compounds | History lead in urine |
| Bulb (mercury) makers | Mercury and its compounds | History mercury in urine |
| Buoy makers | Phosphuretted hydrogen (phosphine) | History |
| Burners (enameling) | Heat lead and its compounds | History lead in urine |
| Burnishers (metals) | Defective illumination antimony and its compounds benzine (naphtha gasoline) carbon tetrachloride sulfuric acid trichlorethylene | History antimony in urine hemogram |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|-------------------------------------|--|---|
| Burrers (needles) | Inorganic dust containing free silica, inorganic dust (except asbestos) containing no free silica | History radiograph of chest, temperature record |
| Burr filers | Inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica | History radiograph of chest temperature record |
| Butchers | Sudden variations in temperature infections (pemphigus erysipeloid tularemia Weil's disease) anthrax septic infections, undulant fever (brucellosis) | History temperature record skin tests with brucellergen (p 317) |
| Button makers | Organic dust inorganic dust (except asbestos) containing no free silica infections acetone chloride of lime formaldehyde | History radiograph of chest temperature record |
| Butyl acetate makers | Butyl alcohol | History |
| Butyl alcohol makers | Butyl alcohol | History |
| Butyl cellosolve makers | Ethylene oxide | History |
| Cable makers | Lead and its compounds | History lead in urine |
| Cable splicers | Dampness carbon monoxide lead and its compounds sulfuretted hydrogen (hydrogen sulfide) turpentine | History lead in urine |
| Cadmium alloy makers | Cadmium | History |
| Cadmium and cadmium compound makers | Arsenuretted hydrogen (arsine) cadmium | History arsenic in urine |
| Cadmium platers | Cadmium See also <i>Electroplaters</i> | History |
| Cadmium vapor lamp workers | Cadmium | History |
| Cannon workers | Abnormalities of air pressure (compressed air—increased atmospheric pressure) sudden variations in temperature dampness defective illumination, carbon dioxide carbon monoxide sulfuretted hydrogen (hydrogen sulfide) | History |
| Calcium cyanamide makers | Heat inorganic dust (except asbestos) containing no free silica calcium cyanamide (cyanamid) | History radiograph of chest temperature record |
| Calenderers (rubber) | Sudden variations in temperature, inorganic dust (except asbestos) containing no free silica | History radiograph of chest temperature record |
| Camphor makers | Amyl acetate aniline and other amino compounds of benzol and its homologues hydrochloric acid zinc | History |
| Candle makers | Acrolein aniline and other amino compounds of benzol and its homologues arsenic and its compounds (except arsenuretted hydrogen) chromium compounds sulfuric acid | History arsenic in urine |
| Cardy makers | Heat sudden variations in temperature | History |
| Carners | Heat sudden variations in temperature dampness septic infections arsenic and its compounds (except arsenuretted hydrogen) carbon dioxide lead and its compounds | History arsenic in urine |
| Can sealers | Benzol (benzene) and its homologues (toluol and xylol) | History hemogram |
| Cap loaders | Mercury and its compounds | History mercury in urine |
| Coppers (window glass) | Heat | History |
| Carbamide makers | Carbon disulfide | History |
| Carbide makers | Heat organic dust inorganic dust (except asbestos) containing no free silica ammoniac carbon monoxid | History radiograph of chest temperature |
| Carbolic acid makers | Benzol (benzene) and its homologues (toluol and xylol) phenol sulfur dioxide sulfuric acid | History routine urine examination hemogram |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|---------------------------------------|---|--|
| Carbonated water makers | Carbon dioxide | History |
| Carbon black workers | Heat organic dust | History radiograph of chest temperature |
| Carbon brush makers | Organic dust, inorganic dust (except asbestos) containing no free silica | History radiograph of chest temperature |
| Carbon-dioxide ice workers | Carbon dioxide | History |
| Carbon-dioxide makers | Carbon dioxide | History |
| Carbon-disulfide makers | Carbon disulfide sulfuretted hydrogen (hydrogen sulfide) | History |
| Carbonic acid makers | Carbon dioxide | History |
| Carbonizers (shoddy) | Organic dust arseniuretted hydrogen (arsine) hydrochloric acid sulfuric acid | History radiograph of chest arsenic in urine |
| Carbon paper makers | Organic dust | History |
| Carbon printers (photography) | Chromium compounds | History |
| Carbon tetrachloride workers | Carbon disulfide carbon tetrachloride phosgene sulfur chloride | History |
| Candlers (asbestos) | Asbestos | History radiograph of chest temperature record |
| Candlers (textiles) | Organic dust | History radiograph of chest temperature record |
| Card grinders (textiles) | Organic dust inorganic dust (except asbestos) containing no free silica | History radiograph of chest temperature record |
| Carpenters | Repeated motion pressure shock etc | History radiograph of chest temperature record |
| Carpet cleaners | Organic dust anthrax | History radiograph of chest temperature record |
| Carpet makers | Organic dust anthrax arsenic and its compounds (except arseniuretted hydrogen) | History radiograph of chest, temperature record arsenic in urine |
| Carroters (felt hats) | Arsenic and its compounds mercury and its compounds nitrous fumes and nitric acid | History arsenic and mercury in urine |
| Cartridge cup washers | Dampness | History |
| Cartridge dippers | Hydrochloric acid nitrous fumes and nitric acid sulfuric acid | History |
| Cartridge felt and wad makers | Dampness | History |
| Cartridge makers | Lead and its compounds mercury and its compounds | History lead and mercury in urine |
| Cartridge shot shell paraffin dippers | Sudden variations in temperature dampness | History |
| Case hardeners | Heat cyanogen compounds | History |
| Casters (metal) | See Foundry and also particular metal | History |
| Casting cleaners (foundry) | Inorganic dust containing free silica in organic dust (except asbestos) containing no free silica See also Acid dippers | History radiograph of chest temperature record |
| Cast rubberers (electroplaters) | Benzine (naphtha gasoline) benzol (benzene) and its homologues (toluol and xylol) | History hemo., ram |
| Catchers (iron and steel) | Heat | History |
| Cattle salesmen | Anthrax undulant fever (brucellosis) | History skin test with brucellergen (p 317) |
| Cellulose formate makers | Formic acid | History |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|--------------------------------------|---|--|
| Cellulose makers | Dampness, sodium hydroxide sulfur dioxide sulfuretted hydrogen sulfuric acid | History |
| Cellulose-products makers | See <i>Rayon pyroxylin plastics lacquers</i> | History |
| Cementers (rubber) | Benzene (naphtha gasoline) benzol (benzene) and its homologues (toluol and xylol) butyl alcohol, carbon disulfide carbon tetrachloride dichlorethylene methanol (methyl alcohol) tetrachlor ethane (acetylene tetrachloride) tri chlorethylene turpentine | History hemogram |
| Cement (Portland) workers | Heat, inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica arsenic and its compounds (except arsenuretted hydrogen) carbon monoxide selenium compounds | History radiograph of chest, temperature record arsenic in urine |
| Cement (rubber plastic, etc.) mixers | Acetone ammonia, amylacetate benzene (naphtha gasoline) benzol (benzene) and its homologues carbon disulfide carbon tetrachloride dioxan (diethylene dioxide) lead and its compounds pyridine sulfur chloride tar and pitch tetrachlorethylene (perchlorethylene) | History lead in urine hemogram |
| Ceramic workers | See <i>Pottery workers</i> | History |
| Chambermen (sulfuric acid) | Sulfur dioxide sulfuric acid | History |
| Charcoal burners | Carbon dioxide carbon monoxide | History radiograph of chest, temperature record |
| Charcoal workers | Organic dust, carbon monoxide | History radiograph of chest, temperature record |
| Charcoal workers (sugar refining) | Heat sudden variations in temperature | History radiograph of chest, temperature record |
| Chargers (furnace) | Heat, inorganic dust (except asbestos) containing no free silica carbon monoxide see also particular metal | History radiograph of chest, temperature record |
| Chargers (smelting and refining) | Heat, inorganic dust (except asbestos) containing no free silica carbon monoxide | History radiograph of chest, temperature record |
| Chasers (teel) | Inorganic dust (except asbestos) containing no free silica | History radiograph of chest temperature record |
| Chauffeurs | Sudden variations in temperature dampness repeated motion pressure shock etc benzene (naphtha gasoline) carbon monoxide | History radiograph of chest, temperature record |
| Chemists (radium research) | X rays radium and other radioactive substances | History hemogram |
| Chimney masons | Carbon monoxide | History |
| Chimney sweepers | Inorganic dust (except asbestos) containing no free silica arsenic and its compounds (except arsenuretted hydrogen) carbon monoxide tar and pitch | History radiograph of chest, temperature record arsenic in urine |
| Chippers | Inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica lead and its compounds | History radiograph of chest, temperature record |
| Chloride-of-lime makers | Chloride of lime chlorine | History |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—*Continued*

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|----------------------------------|---|---|
| Chlorinated diphenyl makers | Chlorinated diphenyls | History |
| Chloroform makers | Inorganic dust (except asbestos) containing no free silica | History radiograph of chest, temperature record |
| Chlorinated naphthalene workers | Chlorinated naphthalenes | History |
| Chlorinated rubber makers | Carbon tetrachloride | History |
| Chlorine compound makers | Hydrochloric acid | History |
| Chlorine makers | Chlorine hydrochloric acid manganese mercury and its compounds | History mercury in urine |
| Chlorodiphenyl makers | Benzol (benzene) and its homologues (toluol and xylol) | History |
| Chrome workers | Chromium compounds | History temperature chart |
| Chromium platers | Chromium compounds see also <i>Electroplaters</i> | History temperature chart |
| Cigar makers | Organic dust fungous infections lead and its compounds nicotine | History radiograph of chest, temperature chart |
| Clay and bisque makers (pottery) | Sudden variations in temperature dampness inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica | History radiograph of chest, temperature chart |
| Clay plug makers (pottery) | Dampness inorganic dust (except asbestos) containing no free silica | History radiograph of chest, temperature chart |
| Clay products workers | See <i>Pottery workers</i> | History radiograph of chest, temperature chart |
| Cleaners (metal) | See <i>Polishers and cleaners (metal)</i> | |
| Clerks | Defective illumination repeated motion pressure shock etc | History |
| Clothes pressers | Carbon dioxide | History |
| Cloth preparers | Heat dampness see also <i>Bleachers</i> | History |
| Cloth singers | Carbon monoxide | History |
| Clutch disk impregnators | Benzol (benzene) and its homologues (toluol and xylol) | History hemogram |
| Coal carbonizers | Sulfuretted hydrogen (hydrogen sulfide) | History radiograph of chest, temperature record |
| Coal miners | See <i>Miners</i> | History radiograph of chest, temperature record |
| Coal passers | Organic dust inorganic dust (except asbestos) containing no free silica | History radiograph of chest, temperature record |
| Coal tar workers | Heat aniline and other amino compounds of benzol and its homologues benzol (benzene) and its homologues (toluol and xylol) carbon monoxide cresol (cresylic acid) cyanogen compounds phenol tar and pitch see also <i>Coke oven workers</i> | History hemogram |
| Cobblers (asbestos) | Asbestos | History radiograph of chest, temperature record |
| Cobblers | Organic dust, anthrax repeated motion pressure shock etc benzol (benzene) and its homologues (toluol and xylol) carbon tetrachloride | History fever chart hemogram |
| Coin makers | Nickel, silver | 1 |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|---|--|--|
| Cane-cutters workers | Hexammonia, benzol (benzene) and its homologs, carbon monoxide, sulfur dioxide, sulfurous hydrogen (hydrogen sulfide), tar and pitch, see also Coal-tar workers | History, hemogram |
| Cold-storage-plant work | See Cold-storage-plant workers | History |
| Cola ("pop") makers | Acetone, methyl alcohol (methyl alcohol), methyl cellosolve (ethylene glycol monomethyl ether) | History |
| Coloring makers | Nitrous fumes and nitric acid | History |
| Coloring-paper workers | Arsenic and its compounds | History, arsenic in urine |
| Coloring (maize) | Chromium compounds | History |
| Coloring (white) of shoes | Lead and its compounds | History, lead in urine |
| Color makers | Flour, pyrogenic dust (except asbestos containing no free silica, ammonia, aniline and other amino compounds of benzol and its homologs, aniline and its compounds, arsenic and its compounds, benzene (naphthalene), benzol (benzene) and its homologs, bromine, cadmium, chlorine, chromium compounds, cobalt, dimethyl sulfoxide, lead and its compounds, manganese, mercury and its compounds, methyl benzoate, methyl chloride, methyl alcohol, organic compounds, sulfur and tellurium, thallium, thallium | History, radiograph of chest, temperature record, arsenic, lead and mercury in urine, hemogram |
| Comb makers | Organic dust, acetone | History |
| Compressors | Defective illuminators, pyrogenic dust (except asbestos, containing no free silica, pyrogenic dust, pressure, shock, etc., aniline and other amino compounds of benzol and its homologs, aniline and its compounds, benzene (naphthalene), lead and its compounds, bromine | History, radiographs of chest, temperature record, hemogram |
| Confounders (rubber) | Lozano dust (except asbestos containing no free silica, aniline and other amino compounds of benzol and its homologs, aniline and its compounds, arsenic and its compounds (except arsenic trioxide), benzene (naphthalene), benzol (benzene) and its homologs, chromium compounds, lead and its compounds | History, radiograph of chest, temperature, arsenic in urine, hemogram |
| Compressed air (cannon) workers | See Cannon workers | History |
| Compressed air (pneumatic tool) workers | See Pneumatic-tool workers | History |
| Concentrating mill workers | Dampest, pyrogenic dust containing free silica, pyrogenic dust (except asbestos containing no free silica, lead and its compounds), manganese, selenium compounds, see also Oil factory—oil workers | History, radiograph of chest, temperature record, lead in urine |
| Coppers (copper) workers | Organic dust, mercury and its compounds | History, mercury in urine |
| Construction workers | See Construction workers | History |
| Cooks | Dampest, pyrogenic dust containing free silica, pyrogenic dust (except asbestos containing no free silica, lead and its compounds, manganese, selenium compounds, see also Oil factory—oil workers | History, radiograph of chest, temperature record |
| Cooks | See Oil factory in temperature, etc., ultraviolet and infra-red radiation, carbon monoxide | History, radiograph of chest, temperature record |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|-------------------------------------|---|--|
| Copper foundries | Arsenic and its compounds copper | History arsenic in urine |
| Copper miners | See <i>Miners</i> | |
| Copper refiners and smelters | Heat antimony and its compounds arsenic and its compounds carbon monoxide copper hydrofluoric acid lead and its compounds manganese selenium compounds sulfur dioxide tellurium compounds | History arsenic and lead in urine hemiogram radiograph of chest temperature record |
| Coppersmiths | Arsenic and its compounds copper | History arsenic in urine |
| Copper (strip) roller mill workers | Acrolein | History |
| Cordage factory workers | Anthrax tar and pitch | History |
| Core makers | Heat inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica carbon monoxide carbon tetrachloride zinc | History radiograph of chest temperature record |
| Cork workers | Organic dust | History radiograph of chest temperature record |
| Corn products workers | Heat, sudden variations in temperature dampness | History radiograph of chest temperature record |
| Cosmetic workers | Arsenic and its compounds mercury and its compounds methyl cellosolve nitrobenzol and other nitro-compounds of benzol and its homologues | History arsenic and mercury in urine hemiogram |
| Cotton mill workers | Heat dampness organic dust, inorganic dust (except asbestos) containing no free silica | History radiograph of chest temperature record |
| Cottonseed-oil workers | Heat | History |
| Cotton twisters | Organic dust repeated motion pressure shock etc | History radiograph of chest temperature record |
| Cranemen (glass industry) | Heat | History |
| Cranemen (iron and steel) | Heat | History |
| Crayon (colored) makers | Chromium compounds lead and its compounds | History lead in urine |
| Creosoting plant workers | Dampness tar and pitch | History radiograph of chest temperature record |
| Cresol soap makers | Cresol | History |
| Cresylic-acid makers | Cresol | History |
| Crucible mixers | Organic dust inorganic dust (except asbestos) containing no free silica | History radiograph of chest temperature record |
| Crucible steel department employees | Heat | History |
| Crushermen (clay and stone) | Inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica | History radiograph of chest temperature record |
| Crushers (asbestos) | Asbestos | History radiograph of chest, temperature record |
| Cupola men (foundries) | Heat carbon dioxide carbon monoxide | History radiograph of chest, temperature record |
| Curers vapor (rubber) | See <i>Vulcanizers</i> | |
| Curriers (tannery) | Organic dust anthrax arsenic and its compounds benzene (naphtha gasoline) | History radiograph of chest temperature record arsenic in urine |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|--|---|--|
| Cut glass workers | Inorganic dust (except asbestos) containing no free silica, arsenic and its compounds lead and its compounds | History radiograph of chest temperature record arsenic and lead in urine |
| Cutlery makers | Inorganic dust containing free silica in organic dust (except asbestos) containing no free silica amyl acetate lead and its compounds | History radiograph of chest lead in urine |
| Cutters (oxyacetylene and other gases) | See Welders | History |
| Cyanamide makers | Heat, inorganic dust (except asbestos) containing no free silica calcium cyanamide (cyanamide) | History radiographs of chest |
| Cyanogen makers | Cyanogen and its compounds mercury and its compounds sulfuretted hydrogen (hydrogen sulfide) | History radiograph of chest, mercury in urine |
| Dairy workers | Anthrax undulant fever (brucellosis) | History radiographs of chest tuberculin reaction skin tests for brucellergin (p 317) |
| Damascening workers | Nitrous fumes and nitric acid | History |
| Dancers | Repeated motion pressure shock etc | History |
| Dead animal handlers | Infections | History tuberculin test skin reaction for brucellergin |
| De-brassers | Nitrous fumes and nitric acid | History |
| Decorators (pottery) | Arsenic and its compounds benzene (naphtha gasoline) benzol (benzene) and its homologues (toluol and xylol) lead and its compounds mercury and its compounds turpentine | History arsenic and mercury in urine hemogram |
| Degreasers | Benzene (naphtha gasoline) benzol (benzene) and its homologues carbon disulfide carbon tetrachloride chlorinated naphthalenes dichloroethyl ether dioxan (d ethylene dioxide) ethylene dichloride methylene chloride (dichloromethane) tetrachlorethane tetrachlorethylene trichlorethylene | History hemogram urine |
| Denatured alcohol workers | See particular denaturant | History |
| Dental workers | Lead and its compounds mercury and its compounds | History lead and mercury in urine |
| Dentists | X rays radium and other radioactive substances mercury and its compounds | History hemogram mercury in urine |
| Depilatory makers | Barium thallium | History hemogram |
| Defining workers | Chlorine | History |
| Detonator fillers | Mercury and its compounds | History mercury in urine |
| Detonator cleaners | Mercury and its compounds | History mercury in urine |
| Detonator packers | Mercury and its compounds | History mercury in urine |
| De il operators (felt hats) | Organic dust mercury and its compounds | History mercury in urine radiographs of chest temperature record |
| Diamond cutters | Organic dust inorganic dust (except asbestos) containing no free silica repeated motion pressure shock etc | History radiographs of chest, temperature record |
| Diamond polishers | Lead and its compounds | History radiograph of chest lead in urine temperature |
| Diatomaceous earth workers | Inorganic dust containing free silica | History radiograph of chest temperature record |
| Dichlorethylene workers | Dichlorethylene | History hemogram |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|---|--|---|
| Digester house workers (paper and pulp) | Heat sudden variations of temperature sulfur dioxide sulfuretted hydrogen | History radiograph of chest temperature chart |
| Dimethyl sulfate workers | Arsenuretted hydrogen dimethyl sulfate methanol (methyl alcohol) nitrous fumes and nitric acid sulfuric acid | History arsenic in urine radiograph of chest temperature record |
| Dinitrobenzol workers | Nitrobenzol and other nitro compounds of benzol and its homologues | History hemogram |
| Dinitrophenol workers | Dinitrophenol | History temperature record examination of lens and eyegrounds |
| Dioxan makers | Dioxan | History |
| Dippers (gun cotton) | Nitrous fumes and nitric acid | History |
| Dippers (rubber) | Benzine (naphtha gasoline) benzol (benzene) and its homologues carbon tetrachloride | History hemogram |
| Dippers | See also <i>Acid dippers</i> | |
| Dish washers | Fungus infections | History |
| Disinfectant makers | Acetaldehyde aniline and other amino compounds of benzol and its homologues arsenic and its compounds benzol (benzene) and its homologues bromine carbon dioxide chloride of lime chlorine cresol cyanogen compounds formaldehyde mercury and its compounds ozone phenol picric acid, sulfur dioxide | History arsenic and mercury in urine hemogram |
| Divers | Compressed air (increased atmospheric pressure) carbon dioxide carbon monoxide | History |
| Doffers (textile) | Heat, dampness organic dust | History radiograph of chest temperature record |
| Dog pound workers | Infections | History hemogram temperature record |
| Dope workers | See <i>Airplane dope makers</i> | |
| Dressers (glass) | Heat | |
| Dresser tenders (textile) | Heat sudden variations in temperature dampness | History radiograph of chest temperature record |
| Driers | Sudden variations in temperature carbon dioxide carbon monoxide | History radiograph of chest temperature record |
| Driers (felt hats) | Sudden variations in temperature methanol (methyl alcohol) | History |
| Driers (lacquer) | Ultraviolet and infra red rays | History hemogram |
| Driers (rubber) | Benzine (naphtha gasoline) benzol (benzene) and its other homologues carbon disulfide | History hemogram |
| Drier workers (foundries) | Carbon monoxide | History |
| Drillers (rock) | Inorganic dust containing free silica in organic dust (except asbestos) containing no free silica | History radiograph of chest temperature record |
| Drop forgers | Heat | History |
| Dry battery workers | See <i>Battery (dry) makers</i> | |
| Dry cleaners | Sudden variations in temperature amyl acetate benzine (naphtha gasoline) benzol (benzene) and its homologues carbon disulfide carbon tetrachloride, dichlorethylene ethylene dichloride methanol (methyl alcohol) oxalic acid tetrachlorethylene tetrachlorethane trichlorethylene turpentine | History hemogram radiograph of chest temperature record |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|---------------------------------------|--|--|
| Drying room workers (miscellaneous) | Sudden variations in temperature carbon dioxide carbon monoxide | History radiograph of chest temperature chart |
| Dye makers | Heat sudden variations in temperature acetaldehyde acetone acridine ammonia aniline and other amino compounds of benzol and its homologues antimony and its compounds, arsenic and its compounds, arseniuretted hydrogen barium benzol (benzene) and its homologues, bromine butyl alcohol carbon dioxide carbon tetrachloride chloride of lime chlorine chromium compounds cresol evanogen compound dimethyl sulfate dinitrophenol diocan ethyl bromide and ethyl chloride formaldehyde formic acid furfural hydrochloric acid lead and its compound manganese mercury and its compounds methanol (methyl alcohol) methyl bromide methyl cellosolve methyl chloride methylene chloride naphthols, nitrobenzol and other nitro compounds of benzol and its homologue nitrous fumes and nitric acid oxalic acid phenol, phenyl hydrazine phosgene picric acid pyridine sodium hydroxide sulfur dioxide sulfuretted hydrogen sulfuric acid thallium trichlorethylene turpentine uranium vanadium | History hemogram temperature record examination of lens and fundus examination of urine for arsenic lead and mercury radiograph of chest |
| Dyers | Sudden variations in temperature acetone ammonia amyl acetate aniline and other amino compounds of benzol and its homologues antimony and its compounds arsenic and its compounds benzene (naphtha gasoline) chromium compounds ethylene dichloride hydrochloric acid lead and its compounds manganese methanol (methyl alcohol) nitrous fumes and nitric acid oxalic acid phenol picric acid, pyridine sulfur chloride titanium oxide uranium vanadium | History hemogram radiograph of chest urine for arsenic and lead temperature chart |
| Electric makers condenser | Chlorinated diphenyls chlorinated naphthalenes | History |
| Electrical makers transformer | Chlorinated diphenyls chlorinated naphthalenes | History |
| Electricians | Ultraviolet and infra red rays ozone | History |
| Electric induction furnace workers | Mercury and its compounds | History mercury in urine |
| Electric linemen | Dampness ultraviolet and infra red rays See also Cable splicers | History |
| Electrode makers | Organic dust tar and pitch | History radiographs of chest temperature chart |
| Electrolytic process (copper) workers | Arseniuretted hydrogen (arsine) | History arsenic in urine |
| Electroplater | Dampness antimony and its compounds arsenic and its compounds benzene (naphtha gasoline) benzol (benzene) and its homologues cadmium carbon disulfide carbon tetrachloride chlorinated naphthalenes chromium com | History hemogram arsenic in urine temperature record lead and mercury in urine |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|---------------------------|---|---|
| | pounds cyanogen compounds formic acid hydrochloric acid hydrofluoric acid lead and its compounds mercury and its compounds nickel nitrous fumes and nitric acid potassium hydroxide sulfuric acid tetrachlorethane trichlorethylene | |
| Electrotypers | Sudden variations in temperature or organic dust inorganic dust (except asbestos) containing no free silica ammonia antimony and its compounds lead and its compounds See also Electroplaters | History radiographs of chest temperature record lead in urine |
| Elevator runners | Repeated motion pressure shock etc | History |
| Embalmers | Formaldehyde mercury and its compounds | History mercury in urine |
| Embalming fluid makers | Mercury and its compounds | History mercury in urine |
| Embossers | Mercury and its compounds | History mercury in urine |
| Embroidery workers | Defective illumination lead and its compounds | History lead in urine refraction |
| Emery wheel makers | Inorganic dust (except asbestos) containing no free silica lead and its compounds | History radiograph of chest temperature record lead in urine |
| Enamelers | Heat dampness inorganic dust containing free silica repeated motion pressure shock etc amyl acetate antimony and its compounds arsenic and its compounds benzene (naphtha gasoline) benzol (benzene) and its homologues carbon disulfide carbon monoxide chromium compounds lead and its compounds manganese nickel tetrachlorethane turpentine | History radiograph of chest hemogram arsenic and lead in urine |
| Enamel makers | Amyl acetate antimony and its compounds arsenic and its compounds barium benzene (naphtha gasoline) benzol (benzene) and its homologues carbon disulfide carbon monoxide chromium compounds hydrochloric acid hydrofluoric acid lead and its compounds manganese, methyl cello-solve nitrous fumes and nitric acid tetrachlorethane turpentine | History hemogram temperature record arsenic in urine |
| Engineers (stationary) | Heat sudden variations in temperature inorganic dust (except asbestos) containing no free silica carbon monoxide | Hemogram radiograph of chest temperature record |
| Engravers | Defective illumination inorganic dust (except asbestos) containing no free silica repeated motion pressure shock etc benzol (benzene) and its homologues copper hydrochloric acid lead and its compounds mercury and its compounds nitrous fumes and nitric acid oxalic acid sodium hydroxide sulfuric acid | Hemogram refraction radiograph of chest hemogram urine for lead and mercury |
| Etchers | Arsenuretted hydrogen, hydrochloric acid hydrofluoric acid, nitrous fumes and nitric acid phenol sulfuric acid | History arsenic in urine |
| Ether makers | Sulfuric acid | History |
| Ethyl benzene makers | Ethyl benzene | History hemogram |
| Ethyl bromide makers | Ethyl bromide and ethyl chloride | History bromine in urine |
| Ethyl chloride makers | Ethyl bromide and ethyl chloride | History bromine in urine |
| Ethylene-dibromide makers | Bromine ethylene dibromide | History bromine in urine |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—*Continued*

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|--------------------------------------|--|---|
| Ethylene-dichloride makers | Ethylene dichloride | History |
| Ethylene oxide makers | Ethylene oxide | History |
| Examiners using fluoroscope or X ray | X rays radium and other radioactive substances | History hemogram |
| Excavation workers | Fungous infections | History |
| Explosives workers | Dampness acetaldehyde acetone ammonia, amyl acetate amyl alcohol aniline and other amino compounds of benzol and its homologues, antimony and its compounds, benzol (benzene) and its homologues, bromine carbon dioxide carbon disulfide chromium compounds cresol, dinitrophenol formaldehyde lead and its compound mercury and its compounds methanol (methyl alcohol) nitrobenzol and other nitro compounds of benzol and its homologues, nitroglycerin nitrous fumes and nitric acid, phenol phosphorus picric acid pyridine sulfuric acid see also particular occupation | History radiograph of chest temperature record hemogram urine for bromine lead mercury examination of lens and fundus |
| Exterminators and fumigators | See <i>Insecticide makers</i> | History |
| Extractor operators (soap) | Sudden variations in temperature dampness | History |
| Extractors (gold and silver) | See <i>Gold and silver refiners and extractors</i> | History |
| Extractors (oils and fats) | Acetone benzine (naphtha gasoline) benzol (benzene) and its homologues carbon disulfide ethylene dichloride tetrachlorethane trichlorethylene | History hemogram |
| Farmers | Infection, anthrax fungous infections undulant fever (brucellosis) arsenic and its compounds, calcium cyanamide lead and its compounds nicotine | History temperature record, tuberculin and brucella tests arsenic and lead in urine |
| Fat renderers | Sudden variations in temperature anthrax acrolein, magnesium ozone sulfuretted hydrogen sulfuric acid | History |
| Feather curers | Organic dust arsenic and its compounds | History arsenic in urine chest radiographs temperature record |
| Feather workers | Organic dust, septic infections aniline and other amino compounds of benzol and its homologues arsenic and its compounds, benzine (naphtha gasoline) benzol (benzene) and its homologues methanol (methyl alcohol) sulfur dioxide turpentine | History chest radiographs temperature record hemogram |
| Felt hat makers | Heat, sudden variations in temperature organic dust, carbon monoxide mercury and its compounds methanol (methyl alcohol) nitrous fumes and nitric acid, sulfuric acid See also particular occupation | History chest radiograph temperature record mercury in urine |
| Felt makers | Heat, anthrax, sulfuretted hydrogen | History chest radiographs, temperature record |
| Ferroalloy workers | Arsenic and its compounds arsenic trihydride, phosphuretted hydrogen | History arsenic in urine |
| Fertilizer makers | Dampness organic dust, inorganic dust containing free silica, inorganic dust (except asbestos) containing no free silica anthrax, septic infections, acro- | History radiograph of chest temperature record, arsenic in urine |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|---------------------------|---|---|
| | pounds cyanogen compounds formic acid hydrochloric acid hydrofluoric acid lead and its compounds mercury and its compounds nickel nitrous fumes and nitric acid potassium hydroxide sulfuric acid tetrachlorethane trichlorethylene | |
| Electrotypers | Sudden variations in temperature or ganic dust inorganic dust (except as bestos) containing no free silica ammonia antimony and its compounds lead and its compounds See also <i>Electroplaters</i> | History radiographs of chest temperature record lead in urine |
| Elevator runners | Repeated motion pressure shock etc | History |
| Embalmers | Formaldehyde mercury and its compounds | History mercury in urine |
| Embalming fluid makers | Mercury and its compounds | History mercury in urine |
| Embossers | Mercury and its compounds | History mercury in urine |
| Embroidery workers | Defective illumination lead and its compounds | History lead in urine refraction |
| Emery wheel makers | Inorganic dust (except asbestos) containing no free silica lead and its compounds | History radiograph of chest temperature record lead in urine |
| Enamelers | Heat dampness inorganic dust containing free silica repeated motion pressure shock etc amyl acetate antimony and its compounds arsenic and its compounds benzene (naphtha gasoline) benzol (benzene) and its homologues carbon disulfide carbon monoxide chromium compounds lead and its compounds manganese nickel tetrachlorethane turpentine | History radiograph of chest hemogram arsenic and lead in urine |
| Enamel makers | Amyl acetate antimony and its compounds arsenic and its compounds barium benzene (naphtha gasoline) benzol (benzene) and its homologues carbon disulfide carbon monoxide chromium compounds hydrochloric acid hydrofluoric acid lead and its compounds manganese methyl cellosolve nitrous fumes and nitric acid tetrachlorethane turpentine | History hemogram temperature record arsenic in urine |
| Engineers (stationary) | Heat, sudden variations in temperature inorganic dust (except asbestos) containing no free silica carbon monoxide | Hemogram radiograph of chest, temperature record |
| Engravers | Defective illumination inorganic dust (except asbestos) containing no free silica repeated motion pressure shock etc benzol (benzene) and its homologues copper hydrochloric acid lead and its compounds mercury and its compounds nitrous fumes and nitric acid oxalic acid sodium hydroxide sulfuric acid | Hemogram refraction radiograph of chest hemogram urine for lead and mercury |
| Etchers | Arsenuretted hydrogen hydrochloric acid hydrofluoric acid nitrous fumes and nitric acid phenol sulfuric acid | History arsenic in urine |
| Ether makers | Sulfuric acid | History |
| Ethyl benzene makers | Ethyl benzene | History hemogram |
| Ethyl bromide makers | Ethyl bromide and ethyl chloride | History bromine in urine |
| Ethyl-chloride makers | Ethyl bromide and ethyl chloride | History bromine in urine |
| Ethylene-dibromide makers | Bromine ethylene dibromide | History bromine in urine |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—*Continued*

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|---|---|---|
| Flint workers | Inorganic dust containing free silica, inorganic dust (except asbestos) containing no free silica | History graph record chest radio-temperature |
| Floor molders | See <i>Molders</i> (foundry) | |
| Floor polish makers | See <i>Polish makers</i> | |
| Flour mill workers | Organic dust, fungous infections | History graph record chest radio-temperature |
| Flue cleaners | Inorganic dust (except asbestos) containing no free silica, carbon monoxide sulfur dioxide tar and pitch | History graph record chest radio-temperature |
| Flue dust recoverers (sulfuric acid mfr.) | Thallium | History hemogram |
| Flush tenders (aluminum) | Dampness | History graph record chest radio-temperature |
| Fly paper makers | Arsenic and its compounds | History arsenic in urine |
| Food irradiators | Ultraviolet and infra red rays | History hemogram |
| Forestry workers | Infections | History temperature record |
| Forgemen | Heat | History |
| Formaldehyde workers | Formaldehyde | History |
| Formers (felt hats) | Organic dust, mercury and its compounds | History chest radio-mercury in urine temperature |
| Formic acid workers | Formic acid oxalic acid | History |
| Foundry workers | Heat, inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica ultraviolet and infra red rays, carbon dioxide carbon monoxide See also particular metal | History graph record chest radio-temperature hemogram |
| Freight handlers | Anthrax | History |
| Frosters (glass and pottery) | Chromium compounds | History |
| Fruit essence makers | See <i>Flavoring extract makers</i> | |
| Fruit preservers | Sulfur dioxide | History |
| Fullers (textiles) | Benzol (benzene) and its homologues carbon tetrachloride dichloroethyl ether tetrachlorethane | History hemogram |
| Fumigant makers | See <i>Insecticide makers</i> | |
| Fumigators and exterminators | See <i>Insecticide makers</i> | |
| Fur causers | Organic dust anthrax | History graph record chest radio-temperature |
| Fur clippers | Organic dust anthrax | History graph record chest radio-temperature |
| Fur cutters | Organic dust anthrax | History graph record chest radio-temperature |
| Fur handlers | Organic dust anthrax mercury and its compounds sulfuretted hydrogen | History graph record chest radio-temperature arsenic and mercury in urine |
| Furnace workers | Heat inorganic dust (except asbestos) containing no free silica ultraviolet and infra red rays carbon dioxide carbon monoxide see also particular metal | History graph record chest radio-temperature hemogram |
| Furniture polishers | Organic dust repeated movement pressure shock etc amyl acetate benzine (naphtha gasoline) chromium compounds methanol petroleum turpentine | History graph hemogram chest radio- |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|--|--|---|
| | lein ammonia arsenic and its compounds arseniuretted hydrogen benzol (benzene) and its homologues calcium cyanamide carbon dioxide cyanogen compounds hydrochloric acid hydro- fluoric acid magnesium manganese nicotine nitrous fumes and nitric acid sulfur dioxide sulfuretted hydrogen sulfuric acid see also <i>Phosphate mill employees</i> | |
| Fiberizers (asbestos) | Asbestos | History chest radio- graph record temperature |
| Fiber workers | Organic dust | History chest radio- graph record temperature |
| Filament makers and fin- ishers (incandescent lamps) | Amyl acetate carbon monoxide meth- anol (methyl alcohol) thallium | History hemogram |
| File cutters | Inorganic dust (except asbestos) contain- ing no free silica lead and its com- pounds | History chest radio- graphs lead in urine temperature chart |
| Filters | Inorganic dust (except asbestos) contain- ing no free silica antimony and its compounds lead and its compounds | History chest radio- graphs lead in urine temperature chart |
| Filling station workers | Benzine (naphtha gasoline) carbon mon- oxide lead and its compounds tetra- ethyl lead | History hemogram |
| Film makers | See <i>Photographic film makers</i> | |
| Filter press workers | Dampness | History chest radio- graph record temperature |
| Finishers (leather) | Organic dust | History chest radio- graph record temperature |
| Fire-extinguisher makers | Carbon dioxide carbon tetrachloric ethyl bromide and ethyl chloride ethylene dibromide methyl bromide | History hemogram |
| Firemen (city) | Heat sudden variations of temperature dampness carbon monoxide phosphorus sulfur dioxide | History chest radio- graph record temperature |
| Firemen (stationary) | Heat sudden variations of temperature inorganic dust (except asbestos) con- taining no free silica ultraviolet and infra red rays carbon monoxide | History chest radio- graph record temperature |
| Fireworks makers | Antimony and its compounds arsenic and its compounds barium manga- nese mercury and its compounds phos- phorus picric acid thallium See also <i>Explosives workers</i> | History arsenic and mer- cury in urine hemo- gram |
| Fishermen | Sudden variations in temperature damp- ness fungus infections tar and pitch | History stool for ova and parasites |
| Fish market workers | Infections | History stool for ova and parasite |
| Hatters (felt hats) | Sudden variations in temperature carbon monoxide | History chest radio- graphs record temperature |
| Flavoring extract makers | Amyl acetate amyl alcohol benzol (ben- zene) and its homologues butyl alco- hol nitrobenzol and other nitro com- pounds of benzol and its homologues | History hemogram |
| Flax rettery workers | Sulfuretted hydrogen | History |
| Flax spinners | Heat, organic dust | History chest radio- temperature |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|---|---|--|
| Glass finishers | Dampness inorganic dust (except asbestos) containing no free silica hydrochloric acid hydrofluoric acid lead and its compounds sulfuric acid | History chest radiograph temperature record lead in urine |
| Glass-furnace workers | Heat, inorganic dust containing free silica, inorganic dust (except asbestos) containing no free silica ultraviolet and infra red rays carbon monoxide see also <i>Glass mixers</i> | History chest radiograph hemogram temperature record |
| Glass mixers | Inorganic dust containing free silica, inorganic dust (except asbestos) containing no free silica antimony and its compounds arsenic and its compounds barium hydrochloric acid lead and its compounds magnesium manganate selenium compounds sodium hydroxide thallium uranium vanadium | History chest radiograph temperature record arsenic and lead in urine hemogram |
| Glass polishers | Lead and its compounds | History lead in urine |
| Glass (safety) makers | Butyl alcohol, methanol tetrachlorethane | History hemogram |
| Glaze dippers (pottery) | Dampness antimony and its compounds arsenic and its compounds chromium compounds hydrochloric acid, lead and its compounds manganese | History chest radiographs temperature record arsenic and lead in urine |
| Glaze mixers (pottery) | Inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica, antimony and its compounds arsenic and its compounds hydrochloric acid, lead and its compounds, manganese | History chest radiographs arsenic and lead in urine temperature record hemogram |
| Glost kiln workers | Sudden variations in temperature carbon monoxide lead and its compounds | History hemogram lead in urine |
| Glove makers (leather preparers) | Dampness organic dust see also <i>Tannery workers</i> | History chest radiograph temperature record |
| Glue workers | Sudden variations in temperature dampness organic dust anthrax septic infections acrol in ammonia benzene (naphtha gasoline) benzol (benzene) and its homologues carbon dioxide carbon disulfide carbon tetrachloride cresol hydrochloric acid nitrobenzol and other nitro compounds of benzol and its homologues sulfur dioxide sulfuretted hydrogen (hydrogen sulfide) sulfuric acid trichlorethylene | History chest radiograph temperature record hemogram |
| Glycerin refiners | Oxalic acid | History |
| Gold beaters | Inorganic dust (except asbestos) containing no free silica repeated motion pressure shock etc | History chest radiographs temperature record |
| Gold and silver refiners and extractors | Inorganic dust (except asbestos) containing no free silica arsenic and its compounds arseniuretted hydrogen bromine chlorine cyanogen compounds formaldehyde hydrofluoric acid lead and its compounds mercury and its compounds sulfur chloride | History chest radiographs temperature record arsenic and bromine in urine lead and mercury in urine hemogram |
| Grain-elevator workers | Organic dust, fungous infections carbon dioxide | History chest radiographs temperature record |
| Granite workers | See <i>Stonecutters</i> | |
| Graphite workers | Heat organic dust inorganic dust (except asbestos) containing no free silica | History chest radiographs temperature record |
| Grinders (colors) | See <i>Color makers</i> | |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|--------------------------|---|---|
| Fur preparers | Organic dust anthrax formaldehyde mercury and its compounds nitrous fumes and nitric acid | History chest radio-graph mercury in urine |
| Fur pullers | Organic dust anthrax | History chest radio-graphs temperature record |
| Fusel oil workers | Amyl alcohol | History |
| Fused quartz workers | Inorganic dust containing free silica | History chest radio-graphs temperature record |
| Calvantr rs | Heat dampness acrolein ammonia arsenic and its compounds arseniuretted hydrogen benzine (naphtha gasoline) hydrochloric acid lead and its compounds nitrous fumes and nitric acid sulfur dioxide sulfonic acid trichlor ethylene zinc | History chest radio-graph hemogram arsenic and lead in urine temperature record |
| Garage workers | Acrolein benzine (naphtha gasoline) carbon monoxide, lead and its compounds tetra-ethyl lead | History chest radio-graphs temperature record lead in urine |
| Garbage workers | Septic infections | History temperature record |
| Gardeners | Undulant fever (brucellosis) arsenic and its compounds calcium cyanamide lead and its compounds nicotine | History skin test for brucellergin, arsenic and lead in urine |
| Gas (illumining) workers | Heat sudden changes in temperature ammonia benzol (benzene) and its homologues arseniuretted hydrogen carbon monoxide cyanogen compounds hydrofluoric acid phenol, sulfuretted hydrogen tar and pitch tri chlorethylene | History chest radio-graph temperature record hemogram arsenic in urine |
| Gasoline blenders | Aniline and other amino compounds of benzol and its homologues benzine (naphtha gasoline) benzol (benzene) and its homologues ethyl benzene ethyl ne dibromide lead and its compounds, nitrobenzol and other nitro compounds of benzol and its homologues tetraethyl lead | History hemogram lead in urine |
| Gasoline engine workers | Acrolein benzine (naphtha gasoline) carbon monoxide | History hemogram |
| Gas purifiers | Ammonia cyanogen compounds phenol sulfuretted hydrogen | History hemogram |
| Gassers (textile) | Carbon monoxide | History |
| Gatherers (glass) | Heat | History |
| Celatine makers | Anthrax acrolein sulfur dioxide | History |
| Germincide makers | See Disinfectant makers | |
| Gild rs | Amyl acetate benzine (naphtha gasoline) benzol (benzene) and its homologues cyanogen compounds mercury and its compounds methanol nitrous fumes and nitric acid pyridine | History hemogram mercury in urine temperature record |
| Glass blowers | Heat, inorganic dust (except asbestos) containing no free silica ultraviolet and infra red rays | History chest radio-graph hemogram temperature |
| Glass colorers | Cadmium chromium compounds cobalt selenium compounds tellurium compounds | History temperature record |
| Glass cutters | Dampness inorganic dust (except asbestos) containing no free silica | History chest radio-graph record |
| Glass etchers | Formaldehyde hydrofluoric acid | History chest radiograph temperature record |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|---|--|---|
| Hunters | Infections | History record temperature |
| Hydraulic construction workers | Dampness | History chest radio-graphs temperature record |
| Hydraulic miners | Dampness | History chest radio-graphs temperature record |
| Hydrochloric-acid makers | Arsenuretted hydrogen hydrochloric acid, sulfuretted hydrogen sulfuric acid | History chest radio-graph temperature record arsenic in urine |
| Hydrocyanic acid makers | Cyanogen compounds sulfuric acid | History chest radio-graph temperature record |
| Hydrofluoric-acid makers | Hydrofluoric acid | History chest radio-graph temperature record |
| Ice (artificial) makers Ice-cream makers | See <i>Artificial ice makers</i> Sudden variations in temperature dampness ammonia carbon dioxide | History chest radio-graphs temperature record |
| Incandescent lamp makers | Amylacetate carbon monoxide lead and its compounds, mercury and its compounds, methanol thallium See also particular occupation | History lead and mercury in urine hemogram |
| Incandescent hardeners | X rays, radium and other radioactive substances, ultraviolet and infra red rays hydrofluoric acid pyridine | History hemogram |
| Ink makers | Ammonia arsenic and its compounds barium benzine (naphtha gasoline) benzol (benzene) and its homologues bromine carbon monoxide carbon tetrachloride chlorine chromium compounds cresol formaldehyde hydrochloric acid, lead and its compounds mercury and its compounds methyl cellosolve nitrobenzol and other nitro compounds of benzol and its homologues oxalic acid potassium hydroxide silver turpentine vanadium | History hemogram arsenic lead and mercury in urine |
| Insecticide makers | Infections arsenic and its compound barium benzine (naphtha gasoline) bromine carbon dioxide carbon disulfide carbon monoxide, carbon tetrachloride cresol, cyanogen compounds dichlorethylene dioxan, ethylene dichloride ethylene oxide formaldehyde lead and its compounds mercury and its compounds methyl bromide methyl formate nicotine nitrous fumes and nitric acid phosphorus sulfur chloride sulfur dioxide tetrachlorethane thallium trichlorethylene turpentine See also <i>Disinfectant makers</i> | History temperature record hemogram arsenic lead and mercury in urine |
| Inspectors using fluoroscope or X ray | X rays radium and other radioactive substances | History hemogram |
| Instrument-dial (luminous) painters | X rays radium and other radioactive substances | History hemogram |
| Insulation (sound) workers | Inorganic dust containing free silica in organic dust (except asbestos) containing no free silica | History chest radio-graphs temperature record |
| Insulators (wire) | Antimony and its compounds arsenic and its compounds benzol (benzene) and its homologues carbon tetrachloride chlorinated diphenyls chlorinated | History hemogram arsenic in urine |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|---|--|--|
| Grinders (metals) | Dampness inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica repeated motion pressure shock etc antimony and its compounds lead and its compounds | History chest radiographs temperature record lead in urine |
| Grinders (rubber) | Organic dust, antimony and its compounds lead and its compounds | History lead in urine chest radiographs temperature record |
| Grinding wheel makers | Inorganic dust containing free silica in organic dust (except asbestos) containing no free silica | History chest radiographs temperature record |
| Grooms | Fungous infections | History temperature record |
| Guncotton dippers | Nitrous fumes and nitric acid sulfuric acid | History chest radiographs temperature record |
| Guncotton pickers | Organic dust | History chest radiographs temperature record |
| Guncotton washers | Dampness | History chest radiographs temperature record |
| Gypsum workers | Sudden variations in temperature inorganic dust (except asbestos) containing no free silica sulfuretted hydrogen | History chest radiographs temperature record |
| Hair workers | Dampness organic dust anthrax septic infections mercury and its compounds | History chest radiographs temperature record mercury in urine |
| Hammermen | Repeated motion pressure shock etc | History |
| Handlers of putrid or decomposing animal products | Septic infections | History chest radiographs temperature record |
| Hardeners (felt hats) | Mercury and its compounds methanol | History mercury in urine |
| Hardeners | See <i>Temperers</i> | |
| Harness makers | Organic dust | History chest radiographs temperature record |
| Heel makers (shoe) | Organic dust | History chest radiographs |
| Hemp workers | Organic dust | History chest radiographs temperature record |
| Hide workers | Fungous infections | History temperature record chest radiograph |
| Horn workers | Organic dust | History chest radiographs temperature record |
| Horse handlers | Glanders and other infections | History chest radiographs temperature record |
| Hospital attendants | X rays radium and other radioactive substances | History chest radiographs temperature record tuberculin reaction, hemogram |
| Hothouse workers | Sudden variations in temperature See also <i>Gardeners</i> | History chest radiographs temperature record |
| Hot rod rollers (iron and steel) | Heat | History |
| Housemaids | Repeated motion pressure shock etc. | History |
| House wreckers | Inorganic dust containing free silica in organic dust (except asbestos) containing no free silica | History chest radiographs temperature |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|---------------------------|---|---|
| | bon tetrachloride cellosolve chlorinated diphenyls chlorinated naphthalenes dioxan, ethylene dichloride formaldehyde formic acid hexanone hexone lead and its compounds methanol methyl cellosolve methylene chloride nitrous fumes and nitric acid pentanone pyridine tetrachlorethane trichlorethylene trorthocresyl phosphate turpentine | |
| Lampblack makers | Organic dust, petroleum phenol | History chest radio-graphs temperature record |
| Lamps (electric) | See <i>Incandescent lamp makers</i> | |
| Lapidaries | Inorganic dust (except asbestos) containing no free silica | History chest radio-graphs temperature record |
| Lard makers | Acrolein | History chest radio-graphs temperature record |
| Lasters (shoe) | Sudden variations in temperature dampness organic dust, methanol | History chest radio-graphs temperature record |
| Lathe turners | Repeated motion pressure shock, etc. | History |
| Laundry workers | Heat, sudden variations in temperature dampness carbon monoxide chloride of lime chlorine formaldehyde ozone | History chest radio-graphs temperature record |
| Lead arsenate makers | Arsenic and its compounds, lead and its compounds | History arsenic and lead in urine |
| Lead barbers | Arsenuretted hydrogen, lead and its compounds | History lead and arsenic in urine |
| Leadfoil makers | Heat, lead and its compounds | History lead in urine |
| Lead miners | Lead and its compounds See also <i>Miners</i> | History chest radio-graphs temperature record |
| Lead-pipe makers | Lead and its compounds | History lead in urine |
| Lead platers (on iron) | Mercury and its compounds | History lead and mercury in urine |
| Lead-salts makers | Lead and its compounds | History lead in urine |
| Lead smelters | Heat, antimony and its compounds, arsenic and its compounds cadmium carbon monoxide lead and its compounds selenium compounds sulfur dioxide tellurium compounds | History chest radio-graphs temperature record, arsenic and lead in urine hemogram |
| Leather workers | Organic dust, anthrax amyl acetate, barium carbon tetrachloride hydrochloric acid, methanol trichlorethylene See also <i>Tannery workers Artificial leather workers</i> | History chest radio-graphs temperature record hemogram |
| Lehr tenders (glass) | Heat | History chest radio-graphs temperature record |
| Letter sorters | Defective illumination repeated motion pressure shock etc. | History refraction |
| Levermen (iron and steel) | Heat | History chest radio-graph temperature record |
| Lifters-over (glass) | Heat | History chest radio-graph temperature record |
| Lime burners | Heat, inorganic dust (except asbestos) containing no free silica arsenuretted hydrogen carbon dioxide carbon monoxide selenium compounds | History chest radio-graph temperature record arsenic in urine |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—*Continued*

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|--|--|--|
| Iodine makers | naphthalenes ethylene dichloride tar and pitch | |
| Iron and steel workers (all departments) | Chlorine Heat inorganic dust (except asbestos) containing no free silica ultraviolet and infra red rays arsenic and its compounds carbon monoxide titanium oxide See also particular occupation and Alloy makers | History History chest radio graphs temperature record arsenic in urine hemogram |
| Ironers | Sudden variations in temperature repeated motion pressure shock etc carbon monoxide | History chest radio-graph temperature record |
| Irradiators (food) | Ultraviolet and infra red rays | History hemogram |
| Japan makers | Sudden variations in temperature arsenic and its compounds benzine (naphtha gasoline) lead and its compounds methanol turpentine | History chest radio graphs temperature record arsenic and lead in urine |
| Japanners | Arsenic and its compounds benzine (naphtha gasoline) lead and its compounds methanol turpentine | History arsenic and lead in urine hemogram |
| Jewelers | Defective illumination inorganic dust (except asbestos) containing no free silica repeated motion, pressure shock etc amyl acetate arseniuretted hydrogen cyanogen compounds hydrochloric acid lead and its compounds mercury and its compounds nitrous fumes and nitric acid sulfuric acid | History refraction, chest radiographs temperature record arsenic lead and mercury in urine |
| Junk (metal) refiners | Heat inorganic dust (except asbestos) containing no free silica lead and its compounds zinc | History chest radio graphs temperature record lead |
| Jute workers | Organic dust, inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica | History chest radio graphs temperature record |
| Kiln tenders | Heat carbon monoxide | History chest radio-graphs temperature record |
| Knitters | Repeated motion pressure shock etc | History |
| Knitting mill workers | Organic dust | History chest radio-graphs temperature record |
| Labelers (paint cans) | Lead and its compounds | History lead in urine |
| Laboratory workers | Carbon monoxide mercury and its compounds | History mercury in urine |
| Laboratory workers (radio research) | X rays radium and other radioactive substances | History hemogram |
| Lace makers | Organic dust | History chest radio-graph temperature record |
| Lacquers | Acetone amyl acetate amyl alcohol arsenic and its compounds benzine (naphtha gasoline) benzol (benzene) and its homologues carbon tetrachloride ethyl benzene ethylene dichloride formic acid lead and its compounds methanol methylene chloride pyridine tetrachlorethane trichlor ethylene triorthocresyl phosphate turpentine | History hemogram arsenic and lead in urine |
| Lacquers makers | Acetaldehyde acetone ammonia amyl acetate amyl alcohol arsenic and its compounds barium benzine (naphtha gasoline) benzol (benzene) and its homologues, butanone butyl alcohol, car | History hemogram temperature record arsenic and lead in urine |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|------------------------------|--|--|
| Manganese-steel makers | Manganese | History chest radio-graphs temperature record |
| Manometer makers | Mercury and its compounds | Mercury in the urine history |
| Manure handlers | Undulant fever (brucellosis) | History temperature record skin test with brucellergen |
| Marble cutters | Inorganic dust (except asbestos) containing no free silica | History chest radio-graphs, temperature record |
| Marblers (glass) | Heat | History chest radio-graphs temperature record |
| Masons | Dampness, inorganic dust containing free silica, inorganic dust (except asbestos) containing no free silica, repeated motion, pressure shock etc | History chest radio-graphs temperature record |
| Massieurs | Infection | History temperature record tuberculin reaction chest |
| Match-factory workers | Dampness, organic dust, inorganic dust (except asbestos) containing no free silica, antimony and its compounds, carbon disulfide, chromium compounds lead and its compounds, manganese phosphorus, potassium hydroxide sulfur-fretted hydrogen | History chest radio-graph temperature record lead in urine |
| Mattress makers | Organic dust, anthrax | History chest radio-graphs, temperature record |
| Meat inspectors | Infections anthrax undulant fever (brucella) | History temperature record skin test for tuberculin and undulant fever with special reference to eosinophils |
| Meat packing employees | See <i>Packing house employees and slaughter houses</i> | |
| Mechanics (gas engines) | Carbon monoxide petroleum | History |
| Melters (foundry) (glass) | Heat | History chest radio-graphs, temperature record |
| Mercerizers | Hydrochloric acid, sodium hydroxide sulfuric acid | History chest radio-graphs temperature record |
| Mercury alloy makers | Mercury and its compounds | History mercury in urine |
| Mercury boiler workers | Mercury and its compounds | History mercury in urine |
| Mercury bronzers | Mercury and its compounds | History mercury in urine |
| Mercury miners | Mercury and its compounds See also <i>Miners</i> | History mercury in urine chest radio-graph temperature |
| Mercury pump workers | Mercury and its compounds | History mercury in urine |
| Mercury-salt workers | Mercury and its compounds | History mercury in urine |
| Mercury-smelters | Heat carbon monoxide mercury and its compounds sulfur dioxide | History mercury in urine |
| Mercury-solder workers | Mercury and its compounds | History mercury in urine |
| Mercury-still workers | Mercury and its compounds | History mercury in urine |
| Mercury-switch makers | Mercury and its compounds | History mercury in urine |
| Mercury vapor lamp makers | Mercury and its compounds | History mercury in urine |
| Metalizers | Cadmium lead and its compounds selenium compounds, zinc | History temperature record lead in urine |
| Metal polishers and cleaners | See <i>Polishers and cleaners (metal)</i> | |
| Metal-polish makers | See <i>Polish makers</i> | |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|-------------------------------|--|---|
| Lime kiln chargers | Inorganic dust (except asbestos) containing no free silica carbon dioxide carbon monoxide | History chest radiograph record temperature |
| Lime pullers (tannery) | Dampness anthrax | History chest radiograph record temperature |
| Lime workers | Inorganic dust (except asbestos) containing no free silica | History chest radiograph record temperature |
| Linen workers | Organic dust | History |
| Linoleum makers | Heat sudden variations in temperature dampness organic dust inorganic dust (except asbestos) containing no free silica acrolein amyl acetate arsenic and its compounds barium benzene (naphtha gasoline) benzol (benzene) and its homologues carbon tetrachloride chromium compounds lead and its compounds manganese methanol sulfuric acid turpentine | History chest radiographs temperature record hemogram arsenic and lead in urine |
| Linotypers | Antimony and its compounds carbon monoxide lead and its compounds | History lead in urine |
| Linseed-oil boilers | Acrolein carbon dioxide lead and its compounds | History lead in urine chest radiographs temperature |
| Litharge workers | Lead and its compounds | History lead in urine |
| Lithographers | Inorganic dust (except asbestos) containing no free silica repeated motion pressure shock etc aniline and other amino compounds of benzol and its homologues arsenic and its compounds benzene (naphtha gasoline) benzol (benzene) and its homologues chromium compounds hydrochloric acid lead and its compounds mercury and its compounds methanol nitrous fumes and nitric acid oxalic acid sulfuric acid tetrachlorethane turpentine | History chest radiographs temperature record hemogram lead and mercury in urine |
| Lithopone makers | Barium cadmium | History temperature record |
| Lithotransfer workers | Lead and its compounds | History lead in urine |
| Longshoremen | Infections (anthrax) manganese | History temperature record |
| Lumbermen | Sudden variations in temperature | History temperature record chest radiograph |
| Luminous dial factory workers | X rays radium and other radioactive substances | History hemogram |
| Luters (zinc smelting) | Heat zinc | History |
| Lye makers | Potassium hydroxide | History |
| Machinists | Repeated motion pressure shock etc | History |
| Magnesium alloy makers | Magnesium | History |
| Mail carriers | Dampness infection | History chest radiographs record temperature |
| Mail sorters | Defective illumination repeated motion pressure shock etc | History refraction |
| Manganese-dioxide workers | Manganese | History |
| Manganese grinders | Manganese | History chest radiographs record temperature |
| Manganese-ore separators | Manganese | History chest radiographs record temperature |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—*Continued*

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|---|---|--|
| | logues antimony and its compounds arsenic and its compounds benzene (naphtha gasoline) benzol (benzene) and its homologues carbon tetra- chloride chromium compounds lead and its compounds | |
| Mixing room workers (miscellaneous) | Organic dust, inorganic dust (except as- bestos) containing no free silica | History chest radio- graph, temperature record |
| Mold breakers (foundry) | Inorganic dust containing free silica, in organic dust (except asbestos) con- taining no free silica | History chest radio- graphs temperature record |
| Mold breakers (pottery) | Carbon monoxide | History chest radio- graphs temperature record |
| Molders (asbestos) | Asbestos | History chest radio- graphs temperature record |
| Monotypers | Antimony and its compounds, carbon monoxide lead and its compounds | History lead in urine |
| Mordanters | Amyl alcohol, antimony and its com- pounds, arsenic and its compounds, benzene (naphtha gasoline) benzol (benzene) and its other homologues, chloride of lime chromium compounds, cyanogen compounds, formic acid ni- trous fumes and nitric acid, vanadium | History hemogram tem- perature record, arsenic in urine |
| Motion-picture film work- ers | Amyl acetate butyl alcohol, carbon mon- oxide tetrachlorethane See also <i>Py- roxylin plastics workers</i> | History hemogram |
| Motion-picture machine operators | Ultraviolet and infra red rays, mercury and its compounds nitrous fumes and nitric acid | History hemogram tem- perature record, chest radiographs |
| Motion picture studio workers and actors | Defective illumination ultraviolet and infra red rays | History hemogram, re- fraction |
| Motormen | Sudden variations in temperature | History chest radio- graphs, temperature record |
| Mottlers (leather) | Amyl acetate methanol | History |
| Muffle tenders | Heat | History chest radio- graphs temperature record |
| Male handlers | Infections | History chest radio- graphs temperature record |
| Muriatic-acid makers | See <i>Hydrochloric acid makers</i> | History |
| Muriatic-acid mixers | See <i>Acid mixers</i> | History |
| Musical-instrument mak- ers | Lead and its compounds | History lead in urine |
| Musicians | Repeated motion, pressure and shock etc. | History chest radio- graphs, temperature record |
| Naphthylamine workers | Aniline and other amino compounds of benzol and its homologues | History hemogram |
| Neon lights lettermakers | Carbon monoxide | History |
| Nickel extractors | Nickel | History temperature rec- ord |
| Nickel platers | Dampness See also <i>Electroplaters</i> | History temperature rec- ord |
| Nickel purification work- ers (Mond process) | Nickel, nickel carbonyl | History temperature rec- ord |
| Nitraniline workers | Aniline and other am. o compounds of benzol and its homologues | History hemogram |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|-----------------------------|--|---|
| Metal turners | Inorganic dust (except asbestos) containing no free silica | History chest radiographs record temperature |
| Metal workers | See particular occupation | |
| Metal washers | Benzene (naphtha gasoline) | History hemogram |
| Methane (synthetic) makers | Carbon monoxide | History |
| Methyl alcohol workers | Acetone carbon monoxide methanol | History |
| Methyl bromide makers | Bromine methanol methyl bromide | History bromine in urine |
| Methyl chloride makers | Hydrochloric acid methanol methyl chloride | History |
| Methyl compounds makers | Methanol | History |
| Methylene chloride workers | Methylene chloride | History |
| Mica strippers or splitters | Inorganic dust (except asbestos) containing no free silica | History chest radiographs record temperature |
| Mica workers | Inorganic dust (except asbestos) containing no free silica | History chest radiographs record temperature |
| Microscopists | Repeated pressure motion shock etc | History refraction |
| Milkers | Fungus infections repeated motion pressure shock etc | History tuberculin and brucellergin skin tests chest radiographs temperature |
| Milk inspectors | Undulant fever (brucellosis) | History chest radiographs temperature record tuberculin and brucellergin reaction |
| Millers | Fungus infections | History chest radiographs temperature record |
| Millinery workers | Aniline and other amino compounds of benzol and its homologues benzene (naphtha gasoline) benzol (benzene) and its homologues methanol turpentine | History hemogram |
| Mineral earth workers | Inorganic dust (except asbestos) containing no free silica | History chest radiographs record temperature |
| Miners | Heat dampness defective illumination inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica asbestos repeated motion pressure shock etc carbon dioxide carbon monoxide manganese nitrous fumes and nitric acid silver sulfuretted hydrogen | History chest radiographs temperature record sputum examination tuberculin reaction |
| Ministry workers | Anthrax | History temperature record |
| Mirror silverers | Sudden variations in temperature dampness acetaldehyde ammonia benzol (benzene) and its homologues cyanogen compounds formaldehyde formic acid, lead and its compounds mercury and its compounds, silver | History chest radiographs temperature record hemogram lead and mercury in urine |
| Mixers (felt hats) | Organic dust, mercury and its compounds | History chest radiographs temperature mercury in urine |
| Mixers (rubber) | Sudden variations in temperature inorganic dust (except asbestos) containing no free silica, aniline and other amino compounds of benzol and its homo- | History chest radiographs temperature record hemogram lead in urine |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—*Continued*

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|--------------------------|--|---|
| Painters (tar) | Tar and pitch | History chest radio-graphs, temperature record |
| Paint makers | Dampness, acetone amyl acetate amyl alcohol aniline and other amino compounds of benzol and its homologues antimony and its compounds arsenic and its compounds barium benzene (naphtha gasoline) benzol (benzene) and its homologues cadmium carbon disulfide carbon tetrachloride chlorinated diphenyl chlorinated naphthalenes chromium compounds, hydrochloric acid lead and its compounds magnesium manganese mercury and its compounds, methanol, phenol pyridine selenium compounds, sodium hydroxide sulfuric acid tar and pitch titanium oxide trichlorethylene, turpentine uranium | History hemogram lead mercury and arsenic in urine |
| Paint remover makers | Benzene (naphtha-gasoline) benzol (benzene) and its homologues butanone carbon tetrachloride, cresol dichloroethyl ether dioxan, furfural, methanol methylene chloride phenol, tetrachloroethane trichlorethylene | History hemogram |
| Paint removers | Inorganic dust (except asbestos) containing no free silica acetone amyl acetate benzene (naphtha gasoline) benzol (benzene) and its homologues butanone carbon tetrachloride cresol dichloroethyl ether lead and its compounds, methylene chloride phenol tetrachloroethane trichlorethylene | History chest radio-graphs, temperature record hemogram lead in urine |
| Pipe heaters (tin plate) | Heat | History chest radio-graphs, temperature record |
| Paper box makers | Repeated motion, pressure shock etc | History |
| Paper glazers | Arsenic and its compounds | History arsenic in urine |
| Paperhangers | Inorganic dust (except asbestos) containing no free silica, arsenic and its compounds, chromium compounds for maldehyde lead and its compounds | History chest radio-graphs, temperature record arsenic in urine lead in urine |
| Paper makers | Heat, sudden variations in temperature fungus infection, ammonia, amyl acetate arseniuretted hydrogen, chlorine chromium compounds formaldehyde hydrochloric acid, hydrofluoric acid lead and its compounds magnesium potassium hydroxide sodium hydroxide, sulfur dioxide sulfuretted hydrogen, sulfuric acid titanium oxide See also particular occupation | History chest radio-graphs, temperature record arsenic in urine |
| Paper money makers | Chromium compounds | History temperature record |
| Paraffin workers | Acetone benzol (benzene) and its homologues carbon disulfide carbon tetrachloride ethylene dichloride, petroleum | History hemogram |
| Parakeet handlers | Psittacosis | History serological reaction, temperature record |
| Pearl-green workers | Arsenic and its compounds | History arsenic in urine |
| Parrot handlers | Psittacosis | History serological test, |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|---|---|--|
| Nitrators | Nitrobenzol and other nitro compounds of benzol and its homologues nitrous fumes and nitric acid sulfuric acid | History hemogram temperature record |
| Nitric acid workers | Ammonia lead and its compounds nitrous fumes and nitric acid sulfuric acid | History temperature record chest radiograph lead in urine |
| Nitrobenzene workers | See <i>Nitrobenzol workers</i> | |
| Nitrobenzol workers | Benzol (benzene) and its homologues nitrobenzol and other nitro compounds of benzol and its homologues nitrous fumes and nitric acid sulfuric acid | History hemogram temperature record |
| Nitrocellulose workers | Acetone amyl acetate amyl alcohol arseniuretted hydrogen benzol (benzene) and its homologues methyl cellosolve nitrous fumes and nitric acid sulfuric acid See also <i>Pyroxylin plastics workers</i> | History hemogram temperature record arsenic in urine |
| Nitroglycerin makers | Arseniuretted hydrogen lead and its compounds nitroglycerin nitrous fumes and nitric acid sulfuric acid | History temperature record arsenic and lead in urine |
| Nitrous-oxide workers | Nitrous fumes and nitric acid | History temperature record |
| Nurses | X rays radium and other radioactive substances | History chest radiographs temperature record tuberculin reaction hemogram |
| Oilcloth makers | See <i>Linoleum makers</i> | |
| Odors | Petroleum | History |
| Oil extractors | See <i>Extractors (oils and fats)</i> | |
| Oil flotation plant workers | Petroleum sulfur dioxide sulfuretted hydrogen, See also <i>Concentrating mill workers</i> | History |
| Oil purifiers | Sulfuric acid | History |
| Oil refiners | See <i>Petroleum refiners</i> | |
| Oil well workers | Petroleum sulfuretted hydrogen | History |
| Open hearth-department workers (iron and steel) | Heat carbon monoxide | History chest radiographs temperature record |
| Ore concentrating mill workers | See <i>Concentrating mill workers</i> | |
| Oxalic acid makers | Cyanogen compounds nitrous fumes and nitric acid oxalic acid potassium hydroxide | History temperature record |
| Oxy acetylene cutters | See <i>Welders</i> | |
| Ozonators | Ozone | History |
| Packing house employees | Heat sudden variations in temperature dampness infections septic infections undulant fever (brucellosis) sulfuretted hydrogen See also <i>Slaughterhouse workers</i> | History chest radiographs temperature record skin test for tuberculin and brucella |
| Painters | Repeated motion, pressure shock etc acetone amyl acetate amyl alcohol aniline and other amino compounds of benzol and its homologues antimony and its compounds arsenic and its compounds barium benzene (naphtha gasoline) benzol (benzene) and its homologues carbon disulfide carbon tetrachloride chromium compounds lead and its compounds manganese mercury and its compounds methanol nitrous fumes and nitric acid trichlor ethylene turpentine | History chest radiographs temperature record hemogram urine for arsenic lead and mercury |
| Painters (luminescent watch and instrument dials) | X rays radium and other radioactive substances | History hemogram |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|--|---|---|
| Phenylhydrazine workers | Phenylhydrazine | History hemogram |
| Phosgene makers | Carbon monoxide chlorine phosgene | History |
| Phosphate extractors | Hydrochloric acid | History |
| Phosphate-mill workers | Sudden variations in temperature dampness inorganic dust (except asbestos) containing no free silica, hydrofluoric acid phosphorus See also <i>Fertilizer makers</i> | History chest radiographs temperature record |
| Phosphoric acid workers | Carbon monoxide phosphuretted by hydrogen | History |
| Phosphor bronze workers | Phosphorus | History chest radiographs temperature record |
| Phosphoretted hydrogen workers | Phosphuretted by hydrogen | History |
| Phosphoric-acid makers | Cyanogen compounds nitrous fumes and nitric acid sulfuric acid | History temperature record chest radiographs |
| Phosphorus-compound makers | Phosphorus, sulfuretted hydrogen | History |
| Phosphorus evaporating machine operators | Sudden variations in temperature dampness phosphorus sulfuric acid | History chest radiographs temperature record |
| Phosphorus extractors | Hydrofluoric acid phosphorus phosphuretted hydrogen | History chest radiographs temperature record |
| Phosphorus (red) makers | Phosphorus phosphuretted hydrogen | History chest radiographs temperature record |
| Photoengravers | Ammonia amyl acetate benzol (benzene) and its homologues chromium compounds methanol nitrous fumes and nitric acid potassium hydroxide | History hemogram temperature record, chest radiographs |
| Photographers | Defective illumination ultraviolet and infra red rays methanol See also <i>Photographic material workers</i> | History refraction hemogram |
| Photographic-film makers | Defective illumination, amyl acetate bromine butyl alcohol nitrous fumes and nitric acid, silver See also <i>Pyroxylin plastics workers</i> | History refraction, temperature record chest radiographs |
| Photographic material workers | Acetaldehyde acetone ammonia, aniline and other amino compounds of benzol and its homologues barium benzol (benzene) and its homologues chlorine, chromium compounds, cresol cyanogen compounds formaldehyde hydrochloric acid mercury and its compounds phenol, picric acid sulfuric acid tellurium compounds trichlor ethylene turpentine uranium vanadium See also <i>Photographic film makers</i> | History hemogram, temperature record urine for mercury |
| Photograph retouchers | Lead and its compounds | History lead in urine |
| Photogravure workers | Chromium compounds, nitrous fumes and nitric acid | History temperature record chest radiographs |
| Physicians | X rays radium and other radioactive substances carbon dioxide mercury and its compounds | History chest radiographs, temperature record, hemogram tuberculin reaction, serology urine |
| Pickers | Heat, dampness arsenuretted hydrogen cyanogen compounds hydrochloric acid hydrofluoric acid nitrous fumes and nitric acid, sulfuric acid | History chest radiograph temperature arsenic in urine |
| Picric-acid makers | Benzol (benzene) and its homologues, nitrous fumes and nitric acid, phenol picric acid, sulfuric acid | History temperature record hemogram |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—*Continued*

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|-----------------------|--|---|
| Patent leather makers | Sudden variations in temperature amyl acetate carbon monoxide lead and its compounds methanol oxalic acid ozone sulfuric acid turpentine | History lead in urine |
| Pavers | Heat repeated motion pressure and shock etc tar and pitch | History chest radio graphs temperature record |
| Pencil makers | Acetone aniline and other amino compounds of benzol and its homologues arsenic and its compounds benzol (benzene) and its homologues chromium compounds pyridine | History hemogram arsenic in urine |
| Perfume makers | Acetone ammonia amyl acetate aniline and other amino compounds of benzol and its homologues benzine (naphtha gasoline) benzol (benzene) and its homologues butyl alcohol carbon tetrachloride cresol dichlorethylene dimethyl sulphate ethyl bromide and ethyl chloride formic acid hydrochloric acid methanol methyl chloride methylene chloride naphthols nitrobenzol and other nitro compounds of benzol and its homologues phenol potassium hydroxide sulfuric acid trichlorethylene | History hemogram urine |
| Petroleum refiners | Heat dampness acetone acrolein ammonia aniline and other amino compounds of benzol and its homologues benzine (naphtha gasoline) benzol (benzene) and its homologues carbon monoxide dichloroethyl ether hydrochloric acid lead and its compounds methylene chloride nitrobenzol and other nitro compounds of benzol and its homologues petroleum sodium hydroxide sulfur dioxide sulfuretted hydrogen sulfuric acid trichlorethylene turpentine | History chest radio graphs temperature record hemogram lead in urine |
| Pewter makers | Antimony and its compounds | History chest radio graphs temperature record |
| Pharmaceutic workers | Organic dust acetone acrolein aniline and other amino compounds of benzol and its homologues antimony and its compounds arsenic and its compounds benzol (benzene) and its homologues bromine calcium cyanamide carbon dioxide carbon tetrachloride chloride of lime dimorphous phenol dioxan ethyl bromide and ethyl chloride ethylene dibromide ethylene dichloride formic acid magnesium manganese mercury and its compounds methyl bromide methyl chloride methylene chloride naphthols nitroglycerin nitrous fumes and nitric acid phenol phenyl hydrazine phosgene phosphorus picric acid potassium hydroxide sodium hydroxide sulfuric acid tellurium compounds tetrachlorethane tetrachlorethylene trichlorethylene turpentine uranium | History chest radio graphs temperature record, hemogram urine for arsenic bromine mercury and lead examination of lens and fundus |
| Phenol makers | Benzol (benzene) and its homologues phenol, sulfuric acid | History hemogram |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|-------------------------------------|--|--|
| Pot room workers (aluminum foundry) | Heat | History chest radio-graphs temperature record |
| Pot setters | Heat | History chest radio-graphs temperature record |
| Pottery workers | Heat, dampness, inorganic dust containing free silica, inorganic dust (except asbestos) containing no free silica, arsenic and its compounds, carbon dioxide carbon monoxide chromium compounds, cobalt, hydrochloric acid hydrofluoric acid, lead and its compounds manganese mercury and its compounds, selenium compounds sulfur dioxide See also particular occupation | History chest radio-graphs temperature record arsenic and mercury in urine |
| Pouncers (felt hats) | Organic dust, inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica | History chest radio-graphs temperature record |
| Pourers (foundry) | Heat | History chest radio-graphs temperature record |
| Powder makers | See <i>Smokeless powder makers</i> | |
| Preparers (tannery) | Dampness, anthrax, septic infections | History chest radio-graphs temperature record |
| Preservative makers and handlers | Formaldehyde | History chest radio-graphs temperature record |
| Pressers | Repeated motion pressure shock etc. carbon monoxide | History chest radio-graphs temperature record |
| Pressmen (oil refining) | Dampness petroleum | History chest radio-graphs temperature record |
| Pressmen (printers) | See <i>Printers</i> | |
| Pressroom workers (rubber) | Sudden variations of temperature aniline and other amino compounds of benzol and its homologues antimony and its compounds arsenic and its compounds benzene (naphtha gasoline) benzol (benzene) and its homologues | History chest radio-graphs temperature record hemogram arsenic in urine |
| Primers (explosives) | Mercury and its compounds | History mercury in urine |
| Printers | Inorganic dust (except asbestos) containing no free silica, aniline and other amino compounds of benzol and its homologues antimony and its compounds arsenic and its compounds benzene (naphtha gasoline) Benzol (benzene) and its homologues, carbon monoxide carbon tetrachloride cyanogen compounds lead and its compounds mercury and its compounds methanol, tetrachlorethylene turpentine | History chest radio-graphs temperature record hemogram arsenic mercury and lead in urine |
| Printers textile | See <i>Textile printers</i> | |
| Puddlers (iron and steel) | Heat, carbon monoxide manganese | History chest radio-graphs temperature record |
| Pullers-out (felt hats) | Heat | History chest radio-graphs temperature record |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|--------------------------------|---|---|
| Pigeon fatteners | Anthrax | History temperature record |
| Pigment makers | See <i>Color makers</i> | |
| Pile drivers | Repeated motion pressure shock etc | History |
| Pipe fitters | Lead and its compounds See also particular liquid piped | History lead in urine |
| Pitch workers | Heat arsenic and its compounds cresol tar and pitch | History chest radio-graphs temperature arsenic urine |
| Planer men (stone) | Inorganic dust containing free silica in organic dust (except asbestos) containing no free silica | History chest radio-graph temperature record |
| Plasterers | Dampness inorganic dust (except asbestos) containing no free silica, an thrax | History chest radio-graph temperature record |
| Plaster-of Paris workers | Inorganic dust (except asbestos) containing no free silica | History chest radio-graph temperature record |
| Platers | See <i>Electroplaters and Metalizers</i> | |
| Platinum extractors | Bromine | History bromine in urine |
| Plumbers | Arsenuretted hydrogen carbon monoxide lead and its compounds See also particular substance piped | History arsenic and lead in urine hemogram |
| Pneumatic tool workers | Inorganic dust (except asbestos) containing no free silica repeated motion pressure shock etc | History chest radio-graphs temperature record |
| Policemen | Dampness carbon monoxide | History chest radio-graphs temperature record |
| Polishers and cleaners (metal) | Defective illumination organic dust in organic dust containing free silica in organic dust (except asbestos) containing no free silica repeated motion pressure shock etc benzine (naphtha gasoline) benzol (benzene) and its homologues cyanogen compounds hydrochloric acid methanol oxalic acid pyridine silver trichlorethylene turpentine | History refraction chest radiograph temperature record hemogram |
| Polish makers | Inorganic dust (except asbestos) containing no free silica amyl acetate aniline and other amino compounds of benzol and its homologues benzine (naphtha gasoline) benzol (benzene) and its homologues carbon tetrachloride cyanogen compounds dioxan, methanol nitrobenzol and other nitro compounds of benzol and its homologues oxalic acid trichlorethylene turpentine | History chest radio-graph temperature record hemogram |
| Porcelain makers | See <i>Pottery workers</i> | |
| Porters | Repeated motion, pressure shock etc | History chest radio-graph temperature record |
| Potassium hydroxide makers | Potassium hydroxide | History |
| Pot fillers (glass) | Heat | History chest radio-graphs temperature record |
| Potlifters (iron and steel) | Heat | History chest radio-graphs temperature record |
| Pot pullers (foundry) | Heat | History chest radio-graphs temperature record |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|---|---|---|
| Red lead workers Roofs (metals) | disulfide hydrochloric acid lead and its compounds mercury and its compounds nitrous fumes and nitric acid phenol sulfuric acid Lead and its compounds Heat arsenic and its compounds arsenic uretted hydrogen carbon monoxide hydrochloric acid lead and its compounds mercury and its compounds nitrous fumes and nitric acid sulfur dioxide sulfuric acid See also particular occupation | History lead in urine History chest radiographs temperature record arsenic lead and mercury in urine |
| Refiners (sugar) Refrigerating plant workers | See <i>Sugar refiners</i> Sudden variations in temperature dampness ammonia carbon dioxide carbon monoxide ozone | History chest radiographs temperature record |
| Refrigerator (mechanical) makers and repairmen | Acrolein ethyl bromide and ethyl chloride methyl bromide methyl chloride methyl formate sulfur dioxide | History chest radiographs temperature record |
| Resins (synthetic) makers | Organic dust acetaldehyde acetone chlorinated diphenyls chlorinated naphthalenes cresol dichloro-ethyl ether formaldehyde furfural methanol methyl cellosolve oxalic acid phenol selenium compounds trichlorethylene vinyl chloride | History chest radiographs temperature record |
| Riveters | Repeated motion, pressure shock etc lead and its compounds | History lead in urine |
| Road repairers | Heat, inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica tar and pitch | History chest radiographs temperature record |
| Roentgenologists | X rays radium and other radioactive substances | History hemogram |
| Roller coverers (cotton mill) | Heat, organic dust | History chest radiographs temperature record |
| Rollers (metals) | Heat | History chest radiographs temperature record |
| Roll setters (iron and steel) | Heat | History chest radiographs temperature record |
| Roll wrenchers (iron and steel) | Heat | History chest radiographs temperature record |
| Roofers | Sudden variations in temperature lead and its compounds tar and pitch | History chest radiographs temperature record lead in urine |
| Roofing material workers | Heat, inorganic dust (except asbestos) containing no free silica asbestos tar and pitch | History chest radiographs temperature record |
| Rope makers | Organic dust tar and pitch | History chest radiographs temperature record |
| Rotogravure workers Roughers (iron and steel) | Benzol (benzene) and its homologues Heat | History hemogram History chest radiographs temperature record |
| Rubber-cement makers Rubber glove makers Rubberized asbestos board makers | See <i>Cement mixers (rubber)</i> Benzine (naphtha gasoline) Benzine (naphtha gasoline) | History hemogram History hemogram |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|---------------------------------|--|--|
| Pulp mill workers | Heat, dampness See also <i>Paper makers</i> | History chest radio- graphs temperature record |
| Putty makers | Inorganic dust (except asbestos) contain- ing no free silica benzene (naphtha gasoline) carbon disulphide lead and its compounds | History chest radio- graphs temperature record hemogram |
| Putty polishers (glass) | Inorganic dust (except asbestos) contain- ing no free silica lead and its com- pounds | History chest radio- graphs temperature record lead in urine |
| Pyridine makers | Pyridine | History hemogram |
| Lyntes burners | Heat inorganic dust (except asbestos) containing no free silica arsenic and its compounds selenium compounds sul- fur dioxide sulfuretted hydrogen | History chest radio- graphs temperature record arsenic in urine |
| Pyroxylin plastics work- ers | Organic dust, acetaldehyde acetone ac- rolein amyl acetate amyl alcohol aniline and other amino compounds of benzol and its homologues arseniuret- ted hydrogen, benzene (naphtha gaso- line) benzol (benzene) and its hom- ologues butyl alcohol carbon mon- oxide carbon tetrachloride cyanogen compounds dichlorethylene, dioxan, ethylene dibromide lead and its com- pounds methanol methyl cellosolve nitrous fumes and nitric acid sulfur- etted hydrogen sulfuric acid tetra- chlorethane, triorthocresyl phosphate | History chest radio- graphs temperature record hemogram ar- senic and lead in urine |
| Quarrymen | Inorganic dust containing free silica in organic dust (except asbestos) contain- ing no free silica repeated motion pressure shock etc | History chest radio- graphs temperature record |
| Quartz workers | Inorganic dust containing free silica | History chest radio- graphs |
| Radioactive paint makers | X rays radium and other radioactive sub- stances | History hemogram |
| Radioactive water mak- ers | X rays radium and other radioactive sub- stances | History hemogram |
| Radiologists | X rays radium and other radioactive sub- stances | History hemogram |
| Radio tube makers | Mercury and its compounds | History mercury in urine |
| Radium miners | X rays radium and other radioactive sub- stances | History hemogram |
| Radium ore reduction workers | X rays radium and other radioactive sub- stances | History hemogram |
| Radium specialists | X rays radium and other radioactive sub- stances | History hemogram |
| Rag workers | Organic dust anthrax septic infections | History chest radio- graphs temperature record |
| Rayon makers | Heat, dampness ammonia amyl acetate, benzene (naphtha gasoline) butyl al- cohol, carbon disulfide chlorinated diphenyls chlorine, cyanogen com- pounds dioxan, formaldehyde hydro- chloric acid methanol, methylene chloride nitrous fumes and nitric acid oxalic acid, sodium hydroxide sulfur- etted hydrogen, sulfuric acid tetra- chlorethane | History hemogram chest radiographs tempera- ture record |
| Reclaimers (rubber) | Aniline and other amino compounds of benzol and its homologues, benzol (benzene) and its homologues carbon | History hemogram lead and mercury in urine |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|---|---|---|
| Sawmill workers | Organic dust | History chest radiograph chest temperature record |
| Sawyers | Repeated motion pressure shock etc | History chest radiograph chest temperature record |
| Sawyers (tone) | Inorganic dust containing free silica in organic dust (except asbestos) containing no free silica | History chest radiograph chest temperature record |
| Scissors sharpeners | Inorganic dust (except asbestos) containing no free silica repeated motion pressure shock etc | History chest radiograph chest temperature record |
| Scourers (belts) | Benzol (benzene) and its homologues | History hemogram |
| Scourers (metals) | Benzine (naphtha gasoline) carbon tetrachloride nitrous fumes and nitric acid sulfuric acid trichlorethylene | History hemogram temperature record |
| Scourers (wood lasts, shoes) | Organic dust | History chest radiograph chest temperature record |
| Scouring powder makers | Inorganic dust containing free silica in organic dust (except asbestos) containing no free silica | History chest radiograph chest temperature record |
| Scrapers (foundry) | Inorganic dust containing free silica in organic dust (except asbestos) containing no free silica | History chest radiograph chest temperature record |
| Screen tenders (pulp mill) | Dampness | History chest radiograph chest temperature record |
| Screen workers (lead and zinc smelting) | Inorganic dust (except asbestos) containing no free silica lead and its compounds | History chest radiograph chest temperature record |
| Scrubwomen | Repeated motion pressure shock etc | History |
| Sealers (incandescent lamps) | Carbon monoxide | History |
| Sealing wax makers | Arsenic and its compounds turpentine | History arsenic in urine |
| Seamstresses | Repeated motion, pressure shock etc | History |
| Selenium refiners | Selenium compounds | History |
| Sewage workers | Dampness, ammonia, benzine (naphtha gasoline) carbon dioxide carbon monoxide sulfuretted hydrogen | History chest radiograph chest temperature record hemogram |
| Sewage purification workers | Chlorine | History hemogram |
| Sewing machine operators | Repeated motion pressure shock etc | History |
| Shade cloth makers | Benzine (naphtha gasoline) benzol (benzene) and its homologues | History hemogram |
| Shale oil workers | See Petroleum refiners | |
| Shavers (felt hats fur tannery) | Dampness, organic dust, anthrax septic infections | History temperature record chest radiograph |
| Shavers | Infection undulant fever (brucellosis) | History skin test temperature record |
| Sheet dip makers | Arsenic and its compounds | History arsenic in urine |
| Sheet metal workers | Lead and its compounds | History lead in urine |
| Shellackers | Amyl acetate benzine (naphtha gasoline) benzol (benzene) and its homologues, butyl alcohol, lead and its compounds methanol turpentine | History hemogram |
| Shellac makers | Ammonium amyl acetate benzine (naphtha gasoline) benzol (benzene) and its homologues, butyl alcohol lead and its compounds methanol, turpentine | History hemogram lead in urine |
| Shell filters | Dinitrophenol, guiterlenol and other nitro compounds of benzol and its homologues, nitroglycerin, picric acid | History hemogram examination of lens and fundus |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|--|---|---|
| Rubber (synthetic) makers | Acetaldehyde amyl alcohol aniline and other amino compounds of benzol and its homologues chlorine chloroprene cresol nitrous fumes and nitric acid sulfur chloride | History hemogram temperature record chest radiographs |
| Rubber tire builders | Benzine (naphtha gasoline) benzol (benzene) and its homologues | History hemogram |
| Rubber workers | Sudden variations in temperature or organic dust inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica acetone aniline and other amino compounds of benzol and its homologues antimony and its compounds arsenic and its compounds barium benzine (naphtha gasoline) benzol (benzene) and its homologues carbon disulfide carbon tetrachloride chromium compounds ethylene dichloride formaldehyde formic acid lead and its compounds magnesium methanol nitrous fumes and nitric acid phenol pyridine sodium hydroxide tellurium compounds tetrachlorethane trichlor ethylene turpentine See also particular occupation | History chest radiographs hemogram temperature record arsenic and lead in urine |
| Sagger makers | Dampness inorganic dust (except asbestos) containing no free silica lead and its compounds | History chest radiographs record temperature |
| Sailors | Sudden variations in temperature repeated motion pressure shock etc carbon monoxide | History chest radiographs record stool for ova and parasites hemogram |
| Salt extractors (coke-oven byproducts) | Ammonia sulfuric acid | History |
| Salt preparers | Heat sudden variations in temperature inorganic dust (except asbestos) containing no free silica | History chest radiographs record temperature |
| Sand blasters | Inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica | History chest radiographs record temperature |
| Sand cutters | Inorganic dust containing free silica | History chest radiographs record temperature |
| Sanders | Inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica | History chest radiographs record temperature |
| Sanding machine operators | Inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica | History chest radiographs record temperature |
| Sandpaperers (enameling) and painting automobiles etc) | Inorganic dust (except asbestos) containing no free silica lead and its compounds | History chest radiographs record lead in urine |
| Sandpaper makers | Inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica | History chest radiographs record temperature |
| Sand pulverizers | Inorganic dust containing free silica | History chest radiographs record temperature |
| Saw filers | Inorganic dust (except asbestos) containing no free silica | History chest radiographs record temperature |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|--------------------------------|---|--|
| Slate workers | Inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica | History chest radiographs, temperature record |
| Slaughter-house workers | Dampness infections anthrax septic infections undulant fever (brucellosis) <i>See also Packing-house workers</i> | History temperature record skin test for tuberculin and brucella, radiographs hemogram ova and parasites |
| Slip makers (pottery) | Dampness, inorganic dust (except asbestos) containing no free silica, lead and its compounds | History chest radiographs, temperature record |
| Slubbers (porcelain enameling) | Lead and its compounds | History lead in urine |
| Smelters | Lead inorganic dust containing free silica, inorganic dust (except asbestos) containing no free silica sulfur dioxide <i>See also particular metal</i> | History chest radiograph, temperature record |
| Smokeless-powder makers | Acetone amyl acetate amyl alcohol, benzol (benzene) and its homologues carbon disulfide nitrobenzol and other nitro compounds of benzol and its homologues nitroglycerin nitrous fumes and nitric acid, phenol picric acid | History hemogram temperature record |
| Smotherers (glass) | Dampness inorganic dust (except asbestos) containing no free silica | History chest radiographs, temperature record |
| Soap (abrasive) workers | Inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica | History chest radiographs, temperature record |
| Soap makers | Sudden variations in temperature dampness organic dust septic infections acrolein, amyl acetate arsenic and its compounds benzene (naphtha gasoline) benzol (benzene) and its homologues carbon tetrachloride dichloroethyl ether ethylene dichloride formaldehyde formic acid hydrochloric acid, manganese methanol nitrobenzol and other nitro compounds of benzol and its homologues, potassium hydroxide, sodium hydroxide sulfuretted hydrogen, sulfuric acid tar and pitch tetrachlorethane tetrachlorethylene trichlorethylene | History chest radiographs, temperature record hemogram arsenic in urine |
| Soda makers | Dampness ammonia, arsenuretted hydrogen, carbon dioxide carbon monoxide chlorine nitrous fumes and nitric acid sulfuretted hydrogen sulfuric acid | History chest radiographs, temperature record arsenic in urine |
| Sodium hydroxide makers | Dampness chlorine sodium hydroxide | History chest radiographs, temperature record |
| Sodium silicate makers | Inorganic dust containing free silica | History chest radiographs, temperature record |
| Sodium sulfide makers | Sulfuretted hydrogen | History |
| Softeners (tannery) | Organic dust | History chest radiographs, temperature record |
| Solderers | Ultraviolet and infra red rays arsenuretted hydrogen, cadmium carbon monoxide cyanogen compounds hydrochloric acid lead and its compounds | History hemogram arsenic and lead in urine |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|-------------------------------|---|--|
| Shepherds | Infections anthrax | History temperature record |
| Sherardizers | Zinc | History temperature record |
| Shingle stainers | Benzine (naphtha gasoline) | History hemogram |
| Shipyard workers | Tar and pitch | History |
| Shoddy workers | Organic dust anthrax septic infections arsenuretted hydrogen chlorine by drochloric acid sulfuric acid | History hemogram temperature record arsenic in urine |
| Shoe dyers | Lead and its compounds nitrobenzol and other nitro compounds of benzol and its homologues | History hemogram lead in urine |
| Shoe factory operatives | Organic dust anthrax acetone amyl acetate benzine (naphtha gasoline) benzol (benzene) and its homologues carbon tetrachloride methanol tetra chloroethane trichloroethylene turpen tine See also particular occupation | History chest radio- graphs temperature record hemogram |
| Shoe finishers | Sudden variations in temperature am monia amyl acetate amyl alcohol benzine (naphtha gasoline) benzol (benzene) and its homologues meth anol | History chest radio- graphs temperature record hemogram |
| Shoe-heel (wood) cov erers | Acetone amyl acetate benzine (naphtha gasoline) benzol (benzene) and its homologues methanol | History hemogram |
| Shooting gallery workers | Mercury and its compounds | History mercury in urine |
| Shot makers | Antimony and its compounds arsenic and its compounds lead and its compounds | History arsenic and lead in urine |
| Shove-in boys (glass) | Heat | History chest radio- graphs temperature record |
| Shifters | Organic dust inorganic dust (except as- bestos) containing no free silica | History chest radio- graphs temperature record |
| Silicon alloy makers | Inorganic dust containing free silica | History chest radio- graphs temperature record |
| Silk weighters | Lead and its compounds tin | History lead in urine |
| Silk workers | Organic dust septic infections | History chest radio- graphs temperature record |
| Silo workers | Carbon dioxide | History |
| Silverers (mirror) | See Mirror silverers | |
| Silver foil makers | Silver | History |
| Silver melters | Sudden variations in temperature carbon monoxide cyanogen compounds silver | History chest radio- graphs temperature record |
| Silver miners | Arsenic and its compounds | History arsenic in urine chest radiographs tem perature record |
| Silver nitrate makers | Silver | History |
| Silver platers | Silver See also Electroplaters | History |
| Silversmiths | Silver | History |
| Singers (cloth) | Carbon monoxide | History |
| Sintering plant workers | Inorganic dust (except asbestos) contain ing no free silica | History chest radio- graphs temperature record |
| Sizers (felt hats) | Heat, mercury and its compounds | History chest radio- graphs temperature mercury in urine |
| Skimmers (glass) | Heat ultraviolet and infra red rays | History hemogram |
| Slag workers | Heat inorganic dust (except asbestos) containing no free silica | History chest rad o- gra hy temperature |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|------------------------------|---|---|
| Stockyard workers | See <i>Slaughter house workers</i> | |
| Stokers | Heat, sudden variations of temperature inorganic dust (except asbestos) containing no free silica ultraviolet and infra red rays carbon monoxide | History chest radio- graphs temperature record hemogram |
| Stone (artificial) makers | Inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica | History chest radio- graphs temperature record |
| Stone cleaners | Hydrofluoric acid oxalic acid | History chest radio- graphs temperature record |
| Stonecutters | Dampness inorganic dust containing free silica, inorganic dust (except asbestos) containing no free silica, repeated motion pressure shock etc | History chest radio- graph temperature record |
| Stone masons | Inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica | History chest radio- graph temperature record |
| Stone workers | Inorganic dust containing free silica, inorganic dust (except asbestos) containing no free silica | History chest radio- graphs temperature record |
| Storage-battery makers | Amyl acetate, antimony and its compounds arsenuretted hydrogen cadmium, carbon monoxide lead and its compounds, mercury and its compounds nickel sulfur dioxide sulfuric acid | History arsenic in urine temperature record lead and mercury in urine |
| Straw cutters | Fungus infections | History chest radio- graphs temperature record |
| Straw hat makers | Sudden variations in temperature or ganic dust, acrolein amyl acetate chloride of lime formaldehyde methanol tetrachlorethane | History chest radio- graphs temperature record |
| Street cleaners | Organic dust, inorganic dust (except asbestos) containing no free silica, septic infection | History chest radio- graphs temperature record |
| Street repairers | Heat, inorganic dust containing free silica, inorganic dust (except asbestos) containing no free silica, tar and pitch | History chest radio- graphs temperature record |
| Submarine workers | Arsenuretted hydrogen carbon dioxide chlorine | History arsenic in urine |
| Sul way construction workers | Inorganic dust containing free silica | History chest radio- graph temperature record |
| Sugar refiners | Heat sudden variations in temperature dampness organic dust, inorganic dust (except asbestos) containing no free silica, ammonia barium carbon dioxide chlorine hydrochloric acid, sulfur dioxide, sulfuretted hydrogen sulfuric acid | History chest radio- graph temperature record |
| Sulfates makers | Sulfuric acid | History |
| Sulfides makers | Sulfuretted hydrogen | History |
| Sulfite cooks (pulp mill) | Heat sudden variations in temperature sulfur dioxide | History chest radio- graphs temperature record |
| Sulfur burners | Heat, inorganic dust (except asbestos) containing no free silica, arsenic and its compounds sulfur dioxide | History chest radio- graphs temperature record arsenic in urine |
| Sulfur chloride makers | Chlorine hydrochloric acid sulfur chloride sulfuric acid | History |
| Sulfur dioxide makers | Carbon monoxide sulfur dioxide | History |
| Sulfurizers (malt and hops) | Sulfur dioxide | History |
| Sulfur extractors | Carbon disulfide | History |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|--------------------------------|--|---|
| Solder makers | Antimony and its compounds cadmium lead and its compounds | History lead in urine |
| Sole stitchers (Blake machine) | Mercury and its compounds | History mercury in urine |
| Soot packers | Organic dust arsenic and its compounds | History arsenic in urine chest radiographs temperature record |
| Spice makers | Organic dust | History chest radiographs temperature record |
| Spinners (asbestos) | Asbestos | History chest radiographs temperature record |
| Spinners (textiles) | Organic dust repeated motion pressure shock, etc | History chest radiographs temperature record |
| Spongers | Heat dampness | History chest radiographs temperature record |
| Sprayers (metals) | See <i>Metallizers</i> | |
| Sprayers (paint) | See <i>Painters</i> | |
| Sprayers (trees) | Arsenic and its compounds cyanogen compounds lead and its compounds | History arsenic and lead in urine |
| Spreaders (rubber works) | Sudden variations in temperature carbon tetrachloride | History temperature record chest radiographs |
| Stablemen | Anthrax, fungus infections undulant fever (brucellosis antrax) | History chest radiographs temperature record skin test with tuberculin and brucella |
| Stamp mill workers | Heat dampness inorganic dust containing free silica inorganic dust (except asbestos) containing no free silica | History chest radiographs temperature record |
| Starch makers | Organic dust, carbon dioxide sulfuretted hydrogen | History chest radiographs temperature record |
| Starters (felt hats) | Heat mercury and its compounds | History mercury in urine |
| Statuary workers | Inorganic dust containing free silica, inorganic dust (except asbestos) containing no free silica | History chest radiographs temperature record |
| Steam fitters | See <i>Pipe fitters</i> | |
| Stearic acid makers | Sudden variations in temperature acrylonitrile | History chest radiographs temperature record |
| Steel alloy makers | See <i>Alloy makers</i> | |
| Steel (chrome) makers | Chromium compounds | History temperature record chest radiograph |
| Steel engravers | See <i>Engravers</i> | |
| Steeple jacks | Carbon monoxide | History |
| Stereotypers | Sudden variations in temperature antimony and its compounds lead and its compounds | History lead in urine chest radiographs temperature chart |
| Stufferers (felt hats) | Mercury and its compounds methanol | History mercury in urine |
| Still (coal tar) cleaners | Heat, benzol (benzene) and its homologues tar and pitch | History chest radiographs temperature record hemogram |
| Stillmen (carbolic acid) | Heat phenol | History chest radiographs temperature record |
| Stillmen operating | Heat. See also particular chemical | |
| Stitchers (shoes) | Methanol | History |
| Stockmen | Undulant fever (brucellosis) | History temperature record chest radiograph skin tests with tuberculin Brucella |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|---|--|--|
| Telegraphers Telephone linemen (trench work) | Repeated motion pressure shock etc. Dampness carbon monoxide See also <i>Cable splicers</i> | History chest radio- graphs temperature record |
| Temperers | Heat, calcium cyanamid carbon monoxide cyanogen compounds, lead and its compounds mercury and its compounds petroleum sulfuric acid | History lead and mercury in urine |
| Tetra thyl lead makers | Bromine lead and its compounds tetra ethyl lead | History lead and bromine in urine |
| Textile (asbestos) workers | Asbestos | History chest radio- graphs temperature record |
| Textile comb makers | Inorganic dust (except asbestos) containing no free silica | History chest radio- graphs, temperature record |
| Textile finishers Textile printers | See particular occupation Heat, sudden variations in temperature amyl acetate aniline and other amino compounds of benzol and its homologues antimony and its compounds arsenic and its compounds cadmium carbon monoxid chlorine chromium compounds cyanogen compounds, formaldehyde hydrochloric acid lead and its compounds manganese mercury and its compounds, methanol, nitrous fumes and nitric acid phenol sulfuric acid turpentine vanadium | History chest radio- graph temperature record hemogram lead and mercury in urine |
| Textile workers | Heat sudden variations in temperature dampness, organic dust. See also particular occupation | History chest radio- graphs temperature record |
| Thallium workers Thermometer makers | Thallium Mercury and its compounds methyl chloride thallium | History History mercury in urine |
| Thread glaziers | Heat, sudden variations in temperature | History chest radio- graph temperature record |
| Tile makers | Heat sudden variations in temperature inorganic dust containing free silica, inorganic dust (except asbestos) containing no free silica, lead and its compounds uranium See also <i>Pottery workers</i> | History chest radio- graph, temperature record lead in urine |
| Tin and lead makers | Heat lead and its compounds | History chest radio- graph temperature record lead in urine |
| Tinners | Heat dampness acrolein ammonia arsenic and its compounds arseniuretted hydrogen hydrochloric acid lead and its compounds | History chest radio- graph, temperature record arsenic and lead in urine |
| Tin plate mill workers Tin recovery workers Tire builders Tobacco denicotinizers Tobacco mixers Tobacco seedling treaters Tobacco workers | See <i>Iron and steel workers</i> Chlorine See <i>Rubber tire builders</i> Ethylene dichloride trichlorethylene Dampness, carbon dioxide Benzol (benzene) and its homologues Organic dust nicotine | History History History History History chest radio- graphs temperature record |
| Tongsman (iron and steel) | Heat | History chest radio- graph temperature record |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—*Continued*

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|-----------------------------------|--|---|
| Sulfuric acid workers | Ammonia arsenic and its compounds arsenuretted hydrogen lead and its compounds nitrous fumes and nitric acid selenium compounds sulfur dioxide sulfuretted hydrogen sulfuric acid vanadium | History arsenic in urine chest radiographs temperature record |
| Sulfur miners | Sulfur dioxide sulfuretted hydrogen | History chest radiographs temperature record |
| Sumackers (tannery) | Dampness anthrax | History chest radiographs temperature record |
| Surgical dressing makers | Phenol | History |
| Tablebands (tannery) | Dampness anthrax | History temperature record chest radiographs |
| Table operatives (iron and steel) | Heat | History temperature record chest radiographs |
| Table turners (enameling) | Sudden variations in temperature inorganic dust (except asbestos) containing no free silica lead and its compounds | History temperature record chest radiographs |
| Tailors | Repeated motion pressure shock, etc carbon monoxide | History temperature record chest radiographs |
| Takers-down (glass) | Heat | History temperature record chest radiographs |
| Talc workers | Inorganic dust (except asbestos) containing no free silica | History temperature record chest radiographs |
| Tallow refiners | Inorganic dust (except asbestos) containing no free silica acrolein carbon disulfide sulfuric acid | History temperature record chest radiographs |
| Tank cleaners | Arsenuretted hydrogen, benzene (naphtha gasoline) benzol (benzene) and its homologues hydrofluoric acid tar and pitch tetraethyl lead See also particular chemical | History hemogram arsenic in urine |
| Tank men | Heat dampness | History radiographs temperature record |
| Tannery workers | Dampness infection septic infections ammonia amyl acetate aniline and other amino compounds of benzol and its homologues arsenic and its compounds benzene (naphtha gasoline) carbon dioxide chloride of lime chromium compounds cyanogen compounds formaldehyde formic acid hydrochloric acid lead and its compounds mercury and its compounds oxalic acid picric acid sodium hydroxide sulfur dioxide sulfuretted hydrogen sulfuric acid | History temperature record chest radiographs hemogram lead and mercury in urine |
| Tappers (air lines) | Tetrachlorethane | History hemogram |
| Tappers (smelting) | Heat See also particular metal | History chest radiographs temperature record |
| Tar (distillery) workers | Heat, arsenic and its compounds cresol tar and pitch See also <i>Coal tar workers</i> | History chest radiographs temperature record arsenic |
| Taxidermists | Organic dust anthrax, septic infections arsenic and its compounds, mercury and its compounds | History chest radiographs temperature record mercury and arsenic in urine |
| Teat gas makers | See <i>Har gas makers</i> | |
| Teazers (glass) | Heat, carbon monoxide | History chest radiographs temperature |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—*Continued*

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|-----------------------------|--|---|
| Vapor cutters Varnishers | See Vulcanizers Acetaldehyde acetone amyl alcohol, aniline and other amino compounds of benzol and its homologues benzine (naphtha gasoline) benzol (benzene) and its homologues butyl alcohol carbon disulfide carbon tetrachloride dichlorethylene ethylene dichloride formic acid lead and its compounds manganese methanol, tetrachlor ethane trichlorethylene turpentine | History hemogram chest radiograph temperature record |
| Varnish makers | Sudden variations in temperature acetaldehyde acetone acrolein, ammonia amyl acetate amyl alcohol aniline and other amino compounds of benzol and its homologues, arsenic and its compounds barium benzine (naphtha gasoline) benzol (benzene) and its homologues, butyl alcohol, carbon disulfide carbon tetrachloride chlorinated diphenyls chlorinated naphthalenes, dioxan, ethylene dichloride formic acid, furfural, hexon lead and its compounds, manganese methanol methyl cellosolve ozone phenol, sodium hydroxide sulfur chloride tetra chloroethane, trichlorethylene turpentine | History hemogram chest radiograph temperature record arsenic in urine |
| Varnish remover makers | Benzine (naphtha-gasoline) benzol (benzene) and its homologues dichloroethyl ether hexanone pentanone tetra chloroethane | History hemogram |
| Varnish removers | Benzine (naphtha gasoline) benzol (benzene) and its homologues dichloroethyl ether hexanone pentanone tetra chloroethane | History hemogram |
| Vatmen | Heat, dampness carbon dioxide | History chest radiograph temperature record |
| Vat varnishers | See Varnishers | |
| Vault workers | Carbon dioxide | History |
| Velvet makers | Heat, arsenic and its compounds | History chest radiograph temperature record arsenic |
| Veterinarians | Infection, anthrax septic infection undulant fever (brucellosis) | History chest radiograph temperature record skin test with tuberculin and brucella hemogram |
| Vignettiers | Hydrochloric acid | History |
| Vin gar workers | Acetaldehyde carbon dioxide | History |
| Vinters | Carbon dioxide | History |
| Vinyl chloride makers | Vinyl chloride | History |
| Vulcanizers | Sudden variations in temperature ammonia, aniline and other amino compounds of benzol and its homologues antimony and its compounds benzene (naphtha gasoline) benzol (benzene) and its homologues carbon dioxide carbon disulphide chromium compounds, methanol, selenium compounds, sulfur chloride sulfur dioxide sulfuretted hydrogen | History chest radiograph temperature record hemogram |
| Vulcanizers (steam) | Heat, dampness | History chest radiograph temperature record |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|--|---|--|
| Toolmakers | Inorganic dust (except asbestos) containing no free silica | History chest radiographs temperature record |
| Top fillers (foundry) | Heat inorganic dust (except asbestos) containing no free silica carbon monoxide | History chest radiographs temperature record |
| Towermen (sulfuric acid) | Arsenuretted hydrogen nitrous fumes and nitric acid sulfur dioxide sulfuric acid see also <i>Sulfuric acid workers</i> | History temperature record arsenic in urine |
| Toy makers | Amyl acetate arsenic and its compounds lead and its compounds | History lead and arsenic in urine |
| Train dispatchers | Defective illumination | History refraction |
| Transfer workers (pottery) | Lead and its compounds turpentine | History lead in urine |
| Transparent wrapping materials workers | Sudden variations in temperature acetone carbon disulphide hydrochloric acid sodium hydroxide sulfuretted hydrogen sulfuric acid | History temperature record chest radiographs |
| Trappers | Infections | History temperature record |
| Treaders (rubber) | Benzene (naphtha gasoline) benzol (benzene) and its homologues | History hemogram |
| Tree sprayers | See <i>Sprayers (trees)</i> | |
| Trichloroethylene workers | Trichloroethylene | |
| Trinitrotoluol makers | Benzol (benzene) and its homologues nitrobenzol and other nitro compounds of benzol and its homologues | History hemogram |
| Tube makers (glass) | Heat | History chest radiographs temperature record |
| Tubulators (incandescent lamps) | Carbon monoxide | History |
| Tumbling barrel workers | Inorganic dust containing free silica in organic dust (except asbestos) containing no free silica | History chest radiographs temperature record |
| Tunnel workers | Compressed air (increased atmospheric pressure) defective illumination in organic dust containing free silica in organic dust (except asbestos) containing no free silica carbon dioxide nitrous fumes and nitric acid sulfuretted hydrogen | History chest radiographs temperature record |
| Turners-out (glass) | Heat | History chest radiographs temperature record |
| Turpentine extractors | Heat, turpentine | History chest radiographs temperature record |
| Type cleaners | Benzene (naphtha gasoline) methanol | History hemogram |
| Type foundries | Antimony and its compounds lead and its compounds | History lead in urine |
| Type melters | Acrolein lead and its compounds | History lead in urine |
| Typesetters | See <i>Compositors</i> | |
| Typists | Repeated motion pressure shock etc | History |
| Ultramarine blue makers | Sulfur dioxide | History |
| Upholsterers | Organic dust, anthrax, methanol | History temperature record chest radiographs |
| Uranium miners | \ rays radium and other radioactive substances uranium | History hemogram |
| Uranium workers | \ rays radium and other radioactive substances uranium | History hemogram |
| Vanadium steel workers | Heat, vanadium | History chest radiographs temperature |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|----------------------------|---|---|
| Wood-alcohol distillers | Acetone carbon monoxide methanol | History |
| Wood-brel workers | Anthrax | History temperature record chest radiograph |
| Wood-last scourers (boes) | Organic dust | History temperature record chest radiograph |
| Wood-polishers | See Furniture polishers | |
| Wood-preserved | Arsenic and its compounds, dinitrophenol, mercury and its compounds, phenol, sulfuric acid, tar and pitch | History arsenic in urine examination of lens and fundus, mercury in urine |
| Wood-stainers | Chromium compounds, lead and its compounds | History lead in urine |
| Wax-workers | Organic dust, benzene (naphtha-gasoline) methanol | History hemogram chest radiograph, temperature |
| Wool-carders | Organic dust, anthrax | History hemogram chest radiograph, temperature |
| Wool-scourers | Sudden variations in temperature dampness, anthrax acetone ammonia | History hemogram chest radiograph, temperature |
| Wool-spinners | Organic dust, anthrax | History hemogram chest radiograph, temperature |
| Wool-workers | Organic dust, anthrax See also particular occupation | History hemogram chest radiograph, temperature |
| X-ray machine makers | Nitrous fumes and nitric acid | History |
| X-ray machine makers | X-rays, radium and other radioactive substances | History hemogram sex glands |
| X-ray photographers | X-rays radium and other radioactive substances | History hemogram sex glands |
| X-ray technicians | X-rays, radium and other radioactive substances | History hemogram sex glands |
| X-ray tube makers | X-rays, radium and other radioactive substances | History hemogram sex glands |
| Yeast makers | Acetaldehyde, carbon dioxide, hydrofluoric acid, sulfuric acid | History |
| Zinc chloride makers | Benzol (benzene) and its homologues, chlorine hydrochloric acid | History hemogram |
| Zinc electrical makers | Mercury and its compounds | History mercury in urine |
| Zinc miners | Cyanogen and its compounds | History |
| Zinc miners | Arsenic and its compounds lead and its compounds, manganese See also Meters | History chest radiograph, temperature record, lead and arsenic in urine |
| Zinc smelters and refiners | Heat, inorganic dust containing free silica, inorganic dust (except asbestos) containing no free silica, antimony and its compounds, arsenic and its compounds, cadmium carbon monoxide lead and its compounds, manganese selenium compounds, sulfur dioxide titanium compounds, zinc | History chest radiograph, temperature record, lead and arsenic in urine |
| Zoological technicians | Undulant fever (Brucella) | History temperature record, skin test with Brucella |

OCCUPATIONAL HAZARDS AND THEIR DIAGNOSTIC FEATURES—Continued

| OCCUPATION | THE HAZARD | DIAGNOSTIC FEATURES |
|---|--|---|
| Wallpaper printers | Heat, sudden variations in temperature arsenic and its compounds, chromium compounds lead and its compounds | History chest radio- graphs, temperature record, arsenic in urine lead in urine |
| Warehouse workers | Anthrax | History temperature rec- ord chest radiographs |
| War gas makers | Arsenuretted hydrogen benzol (benzene) and its homologues, bromine chlorine cyanogen compounds dimethyl sulfate phosgene picric acid sulfur chloride | History hemogram ar- senic in urine chest radiographs, tempera- ture record |
| Warming house employees (gun-cotton) | Sudden variations in temperature | History chest radio- graph, temperature record |
| Washers | Dampness | History chest radio- graph temperature record |
| Washers (metal) | Benzine (naphtha gasoline) See also <i>De greasers</i> | History hemogram |
| Washwomen | Dampness, repeated motion pressure etc | History chest radiograph, temperature record |
| Watch dial (luminous) painters | X rays radium and other radioactive substances | History hemogram |
| Watchmakers | Defective illumination, repeated motion pressure shock etc. | History refraction |
| Water gilders | Mercury and its compounds | History mercury in urine |
| Waterproofers (paper and textiles) | Benzine (naphtha gasoline) benzol (ben- zene) and its homologues, carbon tetra- chloride chromium compounds, for- maldehyde, tar and pitch | History hemogram tem- perature record |
| Water purifiers | Barium chloride of lime chlorine ozone | History |
| Wax makers | Benzol (benzene) and its homologues chlorinated diphenyls, chlorinated naphthalenes, ozone, sulfuric acid, trichlorethane turpentine See also <i>Petroleum</i> | History hemogram |
| Wax-ornament makers | Acrolein, arsenic and its compounds, chromium and its compounds | History arsenic in urine temperature record |
| Weavers | Organic dust, repeated motion, pressure, shock, etc. | History chest radio- graphs, temperature record |
| Weavers (asbestos) | Asbestos | History chest radio- graphs, temperature record |
| Weighers | Organic dust, inorganic dust (except as- bestos) containing no free silica | History chest radio- graphs, temperature record |
| Welders | Heat, ultraviolet and infra red rays, ar- senic and its compounds, benzol (ben- zene) and its homologues, cadmium carbon monoxide chromium and its compounds, copper hydrofluoric acid lead and its compounds, manganese mercury and its compounds, nitrous fumes and nitric acid, ozone phos- phorus, phosphuretted hydrogen, se- lenium compounds, zinc | History chest radio- graphs, temperature record hemogram ar- senic and mercury in urine |
| Well workers | Carbon dioxide | History |
| White lead workers | Carbon dioxide, lead and its compounds | History lead in urine |
| Window-shade makers | Benzine (naphtha gasoline) benzol (ben- zene) and its homologues | History hemogram |
| Wire drawers | Arsenic and its compounds hydrochloric acid, sulfuric acid | History arsenic in urine |
| Wires (incandescent lamps) | Amyl acetate | History |

- Rosenau M J Preventive Medicine and Hygiene 6th ed New York D Appl ton Century Company 1935
- Steghitz E J (ed) Geriatric Medicine The Diagnosis and Management of Disease in the Aging and the Aged Philadelphia W B Saunders Company 1913

SECTION II

INFECTION

- Ash J E and Spitz S Pathology of Tropical Medicine Philadelphia W B Saunders Company 1915
- Blumer G Rickettsial Diseases with Special Reference to those of Importance Along the Atlantic Seaboard Bull New York Acad Med 17:280 1911
- David B D Biologic False Positive Serologic Tests for Syphilis Medicine 23:359 (Dec) 1915
- Foshay L Tularemia Medicine 19:1 (Feb) 1910
- Francis, T. Influenza Bull New York Acad Med 17:269 1911
- Goldberg B (ed) Clinical Tuberculosis 2 vols 4th ed Philadelphia F A Davis Company 1915
- Grace A W Lymphogranuloma Venereum Bull New York Acad Med 17:637 1911
- Harris H J Diagnosis Differential Diagnosis and Treatment of Brucellosis Bull New York Acad Med 19:631 1915
- Herrell W E. Penicillin and Other Antibiotic Agents Philadelphia W B Saunders Company 1915
- Hyman, H T. Massive Dose Chemotherapy by the Intravenous Drop Method Bull New York Acad Med 17:135 1911
- Janeway C A Use of Concentrated Human Serum γ -Globulin in the Prevention and Attenuation of Measles Bull New York Acad Med 21:202 1915
- Jordan E A and Burrows W Textbook of Bacteriology 14th ed Philadelphia W B Saunders Company 1915
- Kolmer J A and Tuft L Clinical Immunology Botherapy and Chemotherapy 3rd ed Philadelphia, W B Saunders Company 1915
- Lancefield R C. Specific Relationship of Cell Composition to Biological Activity of Hemolytic Streptococci The Harvey Lectures Series XXXVI p 251 1940-1941
- Manual of Clinical Mycology National Research Council Division of Medical Sciences Philadelphia W B Saunders Company 1915
- Manual of Tropical Medicine National Research Council, Division of Medical Sciences Philadelphia W B Saunders Company 1915
- McKinley E B The Etiology of Leprosy Medicine 15:377 (Dec) 1934
- Moore J E The Modern Treatment of Syphilis 3rd ed Springfield Ill C C Thomas 1915
- Muckenfuss R S Epidemic Encephalitis Bull New York Acad Med 17:487 1911
- Naper I E L Filariasis Due to Wuchereria Bancrofti Medicine 23:149 (May) 1914
- Paul J R The Clinical Epidemiology of Polomyelitis Medicine 20:495 (Dec) 1911
- Paul J R Polomyelitis Bull New York Acad Med 17:459 1911
- Rivers T M Virus Infections Bull New York Acad Med 17:445 1911
- Simpson W Brucellosis Bull New York Acad Med 17:592 1911
- Smith F G Sulfonamide Therapy in Medical Practice Philadelphia F A Davis Company 1914
- Sutt, E R Diagnosis Prevention and Treatment of Tropical Disease 2 vols 7th ed Philadelphia Blakiston Company 1914
- Stutt G R Clough P W and Clough M C Practical Bacteriology Hematology and Animal Parasitology 9th ed Philadelphia Blakiston Company 1938
- Stokes J H et al Modern Clinical Syphilology 3rd ed Philadelphia W B Saunders Company 1914
- Swift H F Rheumatic Heart Disease Medicine 19:417 (Sept) 1910
- Waksman S A Microbial Antagonisms and Antibiotic Substances New York Commonwealth Fund 1915
- Zinsser H and Bayne-Jones S Textbook of Bacteriology 8th ed New York D Appleton Century Company 1939
- Zinsser H Enders J F and Forthright, R E Immunity Principles and Application in Medicine and Public Health 5th ed New York MacMillan Company 1939

SELECTED REFERENCE LIST

The accompanying list of selected references has been prepared to meet the requirements of physicians practicing in villages towns and smaller cities. Because they do not have the facilities of a fully staffed medical library or of dealers who stock all available medical texts their reading necessarily is limited to current journals and books. The list compiled in the interest of economy and sound medical practice has been chosen from classic and standard text books reviews lectures and articles. Almost without exception these are written in English by prominent medical authorities.

The list is not a complete bibliographic file although the inquiring reader will find a roster of original source material in treatises on each subject. For more complete investigation the physician must consult the *Index Medicus* or the *Quarterly Cumulative Index*. The latter published under the auspices of the American Medical Association appears in installments in the *Journal of the American Medical Association* and also in bound volumes for each year.

SECTION I

GENERAL INTRODUCTION

- Armstrong H G. *Principles and Practice of Aviation Medicine*. 2nd ed. Baltimore: Williams & Wilkins, 1943.
- Best C H, and Taylor N B. *The Physiological Basis of Modern Practice*. 3rd ed. Baltimore: Williams & Wilkins, 1943.
- Boyd W. *A Textbook of Pathology*. 4th ed. Philadelphia: Lea & Febiger, 1944.
- Callander C L. *Surgical Anatomy*. 2nd ed. Philadelphia: W B Saunders Company, 1939.
- Castiglioni A. *History of Medicine*, translated from the Italian and edited by E B Krumpholtz. New York: Alfred A Knopf, 1941.
- Cecil R L (ed). *Textbook of Medicine*. 6th ed. Philadelphia: W B Saunders Company, 1943.
- Cowdry E V. *Problems of Aging*. 2nd ed. Baltimore: Williams & Wilkins, 1943.
- Dorland W A N (ed). *American Illustrated Medical Dictionary*. 20th ed. Philadelphia: W B Saunders Company, 1944.
- Gray H. *Anatomy of the Human Body*. 24th ed. revised and re-edited by W H Lewis. Philadelphia: Lea & Febiger, 1942.
- Howell W H. *Textbook of Physiology*. 15th ed. revised by J Fulton. Philadelphia: W B Saunders Company, 1946.
- Jones T and Shepard W C. *Manual of Surgical Anatomy*. Philadelphia: W B Saunders Company, 1943.
- Karsner H T. *Human Pathology*. 6th ed. Philadelphia: J B Lippincott Company, 1942.
- Loeb L. *Edema*. 271 (Aug.) 1943.
- MacCallum W B. *Textbook of Pathology*. 7th ed. Philadelphia: W B Saunders Company, 1940.
- McCombs R P. *Internal Medicine in General Practice*. Philadelphia: W B Saunders Company, 1943.
- Meakins J C. *The Practice of Medicine*. 2nd ed. St. Louis: C V Mosby Company, 1944.
- Osler Sir William. *The Principles and Practice of Medicine*. 15th ed. (revised by Henry A Christian). New York: D Appleton-Century Company, 1944.

- Graybiel, A. and White P. D. *Electrocardiography in Practice* 2nd ed Philadelphia, W. B. Saunders Company 1946
- Harrison T. R. *Failure of the Circulation* 2nd ed Baltimore Williams & Wilkins 1939
- Leaman, W. G. *Management of the Cardiac Patient*, Philadelphia J. B. Lippincott Company 1943
- Leary T. *Arteriosclerosis* Bull New York Acad Med 1: 587 1941
- Levine S. A. *Clinical Heart Disease* 3rd ed Philadelphia, W. B. Saunders Company 1943
- Levine S. A. *Coronary Thrombosis Its Various Clinical Features* Medicine 8:45 (Sept) 1929
- Libman E. and Friedberg C. *Subacute Bacterial Endocarditis* New York, Oxford University Press, 1941
- Loewe L. *The Combined Use of Penicillin and Heparin in the Treatment of Subacute Bacterial Endocarditis* Canad M. A. J. 5: 1 1943
- Robinson G. C. *The Therapeutic Use of Digitalis* Medicine 11 (May) 1932
- Stroud, W. D. (ed.) *The Diagnosis and Treatment of Cardiovascular Disease* 2 vols Philadelphia, F. A. Davis Company 1943
- Wearn J. T. *Coronary Circulation Morphological and Functional Alterations of* Bull A. M. A. New York Acad Med 1: 754 1941
- White P. D. *Heart Disease* 3rd ed New York, MacMillan Company 1944

SECTION VIII

BLOOD

- Bernstein A. *Infectious Mononucleosis* Medicine 19:33 (Feb) 1940
- Best, C. H. *Heparin and Thrombosis* Bull New York Acad Med 1: 796 1941
- Best, C. H. *Heparin and Thrombosis The Harvey Lectures Series XXXVI* p 66 1940-1941
- Blackfan K. D. and Diamond L. K. *Atlas of the Blood in Children* Illus by C. M. Lester New York, Commonwealth Fund 1944
- Harrop G. A., Jr. *Polycythemia* Medicine 7:291 (Aug) 1928
- Kracke R. R. *Diseases of the Blood and Atlas of Hematology* 2nd ed Philadelphia, J. B. Lippincott Company 1941
- Link, K. P. *The Anticoagulant from Spoiled Sweet Clover Hay* The Harvey Lectures Series XXXIX p 167 1945-1944
- Murphy W. P. *Anemia in Practice* Philadelphia, W. B. Saunders Company 1939
- Whipple A. O. *Medical Surgical Splenopathies* Bull New York Acad Med 1: 174 1939
- Whipple, G. H. and Madison S. C. *Hemoglobin Plasma Protein and Cell Protein—Their Interchange and Construction in Emergencies* Medicine 23:15 (Sept) 1944

SECTION IX

ENDOCRINES

- Albright, F. *Cushing Syndrome: Its Pathological Physiology* The Harvey Lectures Series XXXVIII p 123 1941-1943
- Allen W. M. *Chemical and Physiological Properties and Clinical Uses of the Corpus Luteum Hormone, Progesterone* Bull New York Acad Med 1: 508 1941
- Astwood E. B. *Treatment of Hyperthyroidism with Thiouracil and Thiothiouracil* J. A. M. A. 122:78 (May) 1943
- Cannon, W. B. *The Medulla* Bull New York Acad Med 16:3 1940
- Cramer W. *The Sex Hormones and the Endocrine Balance* Bull New York Acad Med 17:5 1941
- Cushing H. *Papers Relating to the Pituitary Body Hypothalamus and Parasympathetic Nervous System* Springfield, Ill., Thomas 1932
- Daviloff L. M. *Hyperpituitarism and Hypopituitarism* Bull New York Acad Med 16:7 1940
- Geising E. M. K. *The Comparative Anatomy and Pharmacology of the Pituitary Body* The Harvey Lectures Series XXXVII p 269 1941-1942
- Grollman A. *Essentials of Endocrinology* Philadelphia, J. B. Lippincott Company 1940
- Hembecker P. *The Pathogenesis of Cushing's Syndrome* Medicine 23:20 (Sept) 1944
- Hoffman J. *Female Endocrinology* Philadelphia, W. B. Saunders Company 1945
- Howard J. E. *Chemical Physiological and Clinical Aspects of the Androgens* Bull New York Acad Med 17:519 1941

SECTION III

ALLERGY

- Opie E L The Significance of Allergy in Disease Medicine 15 459 (Dec) 1936
 Ratner B Allergy Anaphylaxis and Immunotherapy Baltimore Williams & Wilkins 1913
 Tuft L Clinical Allergy Philadelphia W B Saunders Company 1937
 Vaughan W T The Practice of Allergy St Louis C V Mosby Company 1939

SECTION IV

NEOPLASM

- Lwing J Neoplastic Disease 4th ed Philadelphia W B Saunders Company 1940
 Rhoads C P Recent Studies in the Production of Cancer by Chemical Compounds the Conditioned Deficiency as a Mechanism Bull New York Acad Med 18 53 1912

SECTION V

METABOLISM

- Bean W H Spies T D and Blanckhorn M A Secondary Pellagra Medicine 23 1 (Feb) 1941
 Bodansky M and Bodansky O Biochemistry in Disease New York Macmillan Company 1940
 Cantarow A and Trumper M Clinical Biochemistry 2nd ed Philadelphia, W B Saunders Company 1939
 Dalldorf G Therapeutic Use of Vitamin C Bull New York Acad Med 15 541 1939
 Duncan G G Diseases of Metabolism Philadelphia W B Saunders Company 1940
 Elvehjem C A Biological Significance of Nicotinic Acid Bull New York Acad Med 16 173 1940
 Handbook of Nutrition Council on Foods and Nutrition of the American Medical Association 1913
 Hawk P B and Bergeim O Practical Physiological Chemistry 11th ed Philadelphia Blakiston Company 1944
 McLester J S Nutrition and Diet in Health and Disease 4th ed Philadelphia W B Saunders Company 1943
 Youmans J B Nutritional Deficiency Philadelphia, J B Lippincott Company 1941

SECTION VI

POISONING

- Gonzalez T A Vance M and Halpern M Legal Medicine and Toxicology New York D Appleton Century 1940
 Johnstone R T Occupational Diseases Philadelphia W B Saunders Company 1941
 Lanza, A J and Goldberg J A (eds) Industrial Hygiene New York Oxford University Press 1939
 Manual of Industrial Hygiene and Medical Service in War Industries National Research Council Division of Medical Sciences Committee on Industrial Medicine Philadelphia W B Saunders Company 1943

SECTION VII

CIRCULATION

- Allen E V Thrombo Angitis Obliterans Bull New York Acad Med 18 165 1940
 Chasis H Smith H W and Goldring W Role of the Kidney in the Genesis of Hypertension Bull New York Acad Med 19 419 1943
 Friedlander A Hypotension Medicine 6 143 (May) 1907
 Goldblatt H Experimental Hypertension Induced by Renal Ischemia The Harvey Lectures Series XXXIII p 237 1937-1938
 Goldblatt H and Lewis H A Studies on Experimental Hypertension Bull New York Acad Med 18 459 1940
 Goldring W Management of Hypertension Bull New York Acad Med 19 317 (1913)
 Goldring W and Chasis H Hypertension and Hypertensive Disease New York, Commonwealth Fund, 1944

- Thannhauser S J Neurofibromatosis (von Recklinghausen) and Osteitis Fibrosa Cystica Localisata et Disseminata (von Recklinghausen) A Study of a Common Pathogenesis of Both Diseases Differentiation between Hyperparathyroidism with Generalized Decalcification and Fibrocystic Changes of the Skeleton and Osteitis Fibrosa Cystica Disseminata " *Medicine* 23:10 (May) 1944
- Wechsler I S Textbook of Clinical Neurology 5th ed Philadelphia, W B Saunders Company 1943
- Weiss, E and English E S Psychosomatic Medicine The Clinical Application of Psychopathology to General Medical Problems Philadelphia, W B Saunders Company 1943
- White J and Smithwick, R. The Autonomic Nervous System 2nd ed New York, Mac Millan Company 1941
- Whitehorn, J C. Major Therapeutic Trends in American Psychiatry Bull New York Acad Med 20:446 1944
- Wolff H G. Some Observations on Pain The Harvey Lectures Series XXXIX p 39 1943-44

SECTION XI

EYE

- Duke-Elder W S Textbook of Ophthalmology 4 vols St. Louis C V Mosby Company Vol 1 and 2 1938 Vol 3 1940
- Gifford S R. Textbook of Ophthalmology Philadelphia W B Saunders Company 1945
- May C H Manual of the Diseases of the Eye 18th ed Baltimore William Wood & Company 1913

SECTION XII

DIGESTION

- Alvarez, W C. An Introduction to Gastro-enterology New York, P B Hoeber 1940
- Bacon, H E. Anus Rectum Sigmoid Colon Diagnosis and Treatment, 2nd ed Philadelphia J B Lippincott Company 1941
- Benedict, E B Critical Review of Gastroscopy Bull New York Acad Med 20:179 1944
- Locks H L Gastro-Enterology 3 Vols Philadelphia, W B Saunders Company Vol 1 1943 Vol 2 1944 Vol 3 1946
- Lichtman, S Diseases of the Liver Gallbladder and Bile Ducts Philadelphia Lea & Febiger 1941
- McCall J O., and Wald S S Clinical Dental Roentgenology Philadelphia, W B Saunders Company 1940
- Patek A J., Jr. Dietary Treatment of Laennec's Cirrhosis with Special Reference to Early Stages of the Disease Bull New York Acad Med 19:498 1943
- Porta S A (ed) Diseases of the Digestive System 2nd ed Philadelphia, Lea & Febiger 1944
- Pehfuss M E Indigestion Philadelphia W B Saunders Company 1943
- Thoma, K H Oral Diagnosis Philadelphia W B Saunders Company 1943
- Walters W and Snell A M Diseases of the Gallbladder and Bile Ducts Philadelphia W B Saunders Company 1940

SECTION XIII

RESPIRATION (INCLUDING NOSE, THROAT AND EAR)

- Ballenweber W L Diseases of the Nose Throat and Ear 8th ed Revised by Howard C Ballinger Philadelphia F A Davis Company 1943
- Davidson M Practical Manual of Diseases of the Chest, 2nd ed New York, Oxford University Press 1941
- Jackson C and Jackson C L (eds) Diseases of the Nose Throat, and Ear Philadelphia W B Saunders Company 1943
- Kountz W B and Alexander H L Emphysema Medicine 13:251 (Sept) 1934
- Lederer F L Diseases of the Ear Nose and Throat 3rd ed Philadelphia, F A Davis Company 1943
- Lundsgaard C., and Van Slyke D Cyanosis Medicine 2:1 (Feb) 1923
- Means, J H Dyspnea Medicine 3:309 (Aug) 1924

- Hyman H T The Medical Management of Hyperthyroidism Bull New York Acad Med 16:265 (May) 1940
- Jaffe H L Hyperparathyroidism Bull New York Acad Med 16:291 1940
- Joslin E P Diabetic Manual 7th ed Philadelphia Lea & Febiger 1941
- Lichtwitz L Hypothalamic Pituitary Syndromes Bull New York Acad Med 15:73 1939
- Loeb R. Adrenal Cortex and Electrolyte Behavior Bull New York Acad Med 18:663 1942
- Loeb R F The Adrenal Cortex and Electrolyte Behavior The Harvey Lectures Series XXXVII p 100 1941-1942
- Loeb R Adrenal Gland Insufficiency Bull New York Acad Med 16:347 1940
- Marine D. Etiology and Prevention of Simple Goiter Medicine 2:453 (Nov) 1924
- Marine D Iodine in the Treatment of Diseases of the Thyroid Gland Medicine 6:197 (Feb) 1927
- Marine D Physiology and Principal Interrelations of the Thyroid Gland Bull New York Acad Med 14:790 (Dec) 1939
- Means J H Hypothyroidism Diagnosis and Treatment Bull New York Acad Med 16:14 1940
- Moore C R Physiology of the Testes and Therapeutic Application of Male Hormone Bull New York Acad Med 16:135 1940
- Stadie W C Intermediary Metabolism in Diabetes Mellitus The Harvey Lectures Series XXXVII p 129 1941-1942
- Thorne G The Clinical Use of Extracts from Adrenal Cortex J A M A 125:10 (May) 1944
- Tolstoi E Dietary Control in Diabetes J A M A 115:454 1940
- Werner A A Endocrinology Clinical Application and Treatment 2nd ed Philadelphia, Lea & Febiger 1942
- Wilder R M. A Primer for Diabetic Patients 7th ed Philadelphia W B Saunders Company 1941
- Wilder R M Clinical Diabetes Mellitus and Hyperinsulinism Philadelphia W B Saunders Company 1940
- Williams R H and Bissell G W Thiouracil in the Treatment of Thyrotoxicosis New England J Med 209:97 (July) 1943

SECTION X

NEUROLOGY AND PSYCHIATRY

- Alexander F G Indications for Psychoanalytic Therapy Bull New York Acad Med 20:319 1944
- Haymaker W and Woodhall B Peripheral Nerve Injuries Philadelphia W B Saunders Company 1945
- Horrax G Diagnosis and Prognosis of Brain Tumors Bull New York Acad Med 19:125 1943
- Hyman H T The Value of Psychoanalysis as a Therapeutic Procedure J A M A 107:326 (Aug) 1936
- Kalinowsky L B Experience with Electric Therapy in Various Types of Psychiatric Patients Bull New York Acad Med 20:485 1944
- Kubie L S Basis of a Classification of Disorders from the Psychosomatic Standpoint Bull New York Acad Med 20:46 1944
- Kubie L S Nature of Psychotherapy Bull New York Acad Med 19:183 1943
- Lennox W G. Prevention and Treatment of Convulsive Disorders Bull New York Acad Med 19:47 1943
- Lewis N D C Present Status of Shock Therapy of Mental Disorders Bull New York Acad Med 19:227 1943
- Lewis Sir Thomas Pain New York MacMillan Company 1942
- Neurosurgery and Thoracic Surgery National Research Council Philadelphia W B Saunders Company 1943
- Pacella B L. Convulsive Shock Therapy Bull New York Acad Med 20:572 1944
- Ranson S W Anatomy of the Nervous System 7th ed Philadelphia W B Saunders Company 1943
- Solomon H C and Yakovlev P I. Manual of Military Neuropsychiatry Philadelphia W B Saunders Company 1944
- Strecker E A. and Ebaugh F G Practical Clinical Psychiatry 5th ed Philadelphia Blakiston Company 1940

- Mitchell A G and Nelson W F Textbook of Pediatrics 4th ed Philadelphia W B Saunders Company 1945
- Holt L F and Howland J Diseases of Infancy and Childhood 11th ed Revised by L E Holt, Jr and R McIntosh New York D Appleton Century Company 1943
- Lester C W Acute Surgical Conditions of the Abdomen in Children Bull New York Acad Med 1944
- McIntosh R Disorders of the Digestive System Leading to Vitamin Deficiency States in Infants and Children Bull New York Acad Med 1944

SECTION XIX

SKELETAL AND LOCOMOTOR SYSTEMS

- Conroe B I Arthritis and Allied Conditions 3rd ed Philadelphia Lea & Febiger 1944
- Hawser F D W Diseases of the Foot Philadelphia W B Saunders Company 1939
- Key J A and Connell H E Fractures Dislocations and Sprains 3rd ed St Louis C V Mosby Company 1942
- Lewis P The Foot and Ankle 2nd ed Philadelphia Lea & Febiger 1941
- Moorhead J J Traumatic Surgery Philadelphia, W B Saunders Company 1945
- The Principles and Outline of Fracture Treatment Bull Am Coll Surgeons (Mar) 1931
- Sculler C L The Treatment of Fractures 11th ed Philadelphia W B Saunders Company 1938
- Shaar C M and Kretz T P Manual on Treatment of Fractures Philadelphia W B Saunders Company 1943
- Steinbrocker O Arthritis in Modern Practice Philadelphia W B Saunders Company 1941
- Watson-Jones R Fractures and Other Bone Joint Injuries 2 Vols 3rd ed Baltimore Williams & Wilkins 1943

SECTION XX

SUPPLEMENTARY SYSTEM

- Andreas C Diseases of the Skin 2nd ed Philadelphia W B Saunders Company 1939
- Manual of Clinical Mycology National Research Council Division of Medical Sciences Philadelphia W B Saunders Company 1945
- Manual of Dermatology National Research Council Philadelphia W B Saunders Company 1942
- Manual of Tropical Medicine National Research Council Division of Medical Sciences Philadelphia W B Saunders Company 1945
- Sulzberger M B and Wolf J Dermatologic Therapy in General Practice 2nd ed Chicago Year Book Publishers 1942
- Sutton R L and Sutton R L Jr Diseases of the Skin 10th ed St Louis C V Mosby Company 1939

SECTION XXI

PHYSICAL DIAGNOSIS

- Cabot R C Differential Diagnosis 2 Vols 4th ed Philadelphia W B Saunders Company 1919
- Cabot R C and Adams F S Physical Diagnosis 13th ed Baltimore Williams & Wilkins Company 1942
- French H and Doullwaite A H Index of Differential Diagnosis of Main Symptoms 6th ed Baltimore Williams & Wilkins 1945
- Mayer R H Physical Diagnosis 3rd ed Philadelphia W B Saunders Company 1945
- Meakins J C Symptoms in Diagnosis Baltimore Williams and Wilkins Company 1941
- Pullen R I (ed) Medical Diagnosis Applied Physical Diagnosis Philadelphia W B Saunders Company 1944
- Yater W M Symptom Diagnosis 4th ed New York D Appleton Century Company 1942

SECTION XXII

LABORATORY METHODS

- Craig C F and Faust E C Clinical Parasitology 3rd ed Philadelphia Lea & Febiger 1943
- Golden R (ed) Diagnostic Roentgenology 2 Vols New York Thomas Nelson & Sons 1943

Norris G W and Landis H R M Diseases of the Chest 6th ed Philadelphia W B Saunders Company 1938

SECTION XIV

URINARY SYSTEM

- Fishberg A Hypertension and Nephritis 4th ed Philadelphia Lea & Febiger 1939
 Herman L The Practice of Urology Philadelphia W B Saunders Company 1938
 Hinman F The Principles and Practice of Urology Philadelphia W B Saunders Company 1935
 Lester L Nephrosis Medicine 10 135 (May) 1931
 Newburgh I H The Etiology of Nephritis Medicine 2 77 (Feb) 1923
 Jelouze P S Office Urology Philadelphia W B Saunders Company 1940
 Smith H W Physiology of the Renal Circulation The Harvey Society Lectures Series XXXV p 166 1939-1940

SECTION XV

MALE REPRODUCTIVE SYSTEM

- Hotchkiss R S Testicular Biopsy in the Diagnosis and Treatment of Sterility in the Male Bull New York Acad Med 18 600 1942
 Moore C R Physiology of the Testes and Therapeutic Application of Male Hormone Bull New York Acad Med 16 195 1940
 Werner A A The Male Climacteric J A M A 112 1441 (April) 1939

SECTION XVI

FEMALE REPRODUCTIVE SYSTEM

- Crossen H S and Crossen P J Diseases of Women 9th ed St Louis C V Mosby Company 1941
 Curtis A J Textbook of Gynecology 5th ed Philadelphia W B Saunders Company 1946
 Hoffman J Female Endocrinology Philadelphia W B Saunders Company 1941
 Janney J C Medical Gynecology Philadelphia W B Saunders Company 1941
 Rubin I C Diagnosis of the Etiological Factors in Female Sterility Bull New York Acad Med 18 537 1942
 Smith P F Physiology of the Ovaries Bull New York Acad Med 16 153 1940
 Wharton L R Gynecology With a Section on Female Urology Philadelphia W B Saunders Company 1945

SECTION XVII

OBSTETRICS

- Beck A Textbook of Obstetrics Baltimore Williams & Wilkins 1941
 DeLee J B and Greenhill J P Principles and Practice of Obstetrics 8th ed Philadelphia W B Saunders Company 1943
 Dexter L and Weiss S Pre-eclamptic and Eclamptic Toxemia of Pregnancy Baltimore Williams & Wilkins 1941
 Edwards W B and Hingston R A Present Status of Continuous Caudal Analgesia in Obstetrics Bull New York Acad Med 19 507 1943
 Stander H Obstetrics New York D Appleton Century Company 1941
 Stander H Toxemias of Pregnancy Medicine 81 (Feb) 1929
 Studdiford W E Puerperal Infection Bull New York Acad Med 17 67 1941
 Williams J W Obstetrics 8th ed Revised by H J Stander New York D Appleton Century Company 1941
 Zabriskie L and Eastman N J Nurses Handbook of Obstetrics Philadelphia J B Lippincott Company 1943

SECTION XVIII

PEDIATRICS

- Da Costa W C The Complete Pediatrics 4th ed Durham N C Duke University Press 1945

REHABILITATION AND CONVALESCENT CARE

THE APPLICATION OF MILITARY EXPERIENCE TO CIVILIAN PRACTICE

By

HOWARD A. RUSK, M.D.*

The medical aphorism that states that the more the healer knows about his patient's disease the easier it is for him to lose touch with the patient is nowhere better illustrated than in the field of rehabilitation and convalescent care. Great advances have been made in preventive and curative medicine and surgery but the third phase of medical care—rehabilitation and convalescent care—has been sadly neglected. We have often been so busy treating diseases that we have forgotten to treat patients. In our search for new drugs, diagnostic procedures, surgical techniques and laboratory methods we have too frequently overlooked the effect of disease upon the individual in terms of its economic, vocational, social and personal effects.

THE ART OF MEDICINE IN THE MILITARY PROGRAM

In applying the science of medicine we have sometimes forgotten the art as particularly evidenced in the case of military hospitalization in the early days of the war. In those dark and uncertain days every man hour of training in the Air Forces was unbelievably important. Our radio schools, mechanic schools and flying schools worked around the clock 24 hours a day seven days a week. It was necessary in this critical period for each man to pull his share of the load. Yet in our military hospitals thousands of men sat around merely waiting for time and nature to complete their convalescence. Their definitive treatment was over and now they had nothing to do but sit and wait. That waiting turned into long periods of boredom in which they read comic books and adventure stories and did nothing to aid either themselves or the Army Air Forces. Their doctors devoted attention to the new patients and forgot the convalescents who waited often for a long time. Convalescence is a longer period in military medicine than in civilian practice for in the Army a man is either sick in hospital or on active duty; there is no in-between period as in civilian practice where the doctor can tell the patient to go home and take it easy for a few days and don't go to work until I see you two weeks from today. Full duty is arduous, strenuous activity and before the soldier patient is discharged from the hospital he must be ready physically for these long and strenuous hours of activity.

Army Air Forces Convalescent Training Program—It was to convert this

Dr. Rusk conceived and organized the Army Air Forces Convalescent and Rehabilitation Program. He is consultant in physical rehabilitation to the Baruch Committee on Physical Medicine.

- Sante L. Manual of Roentgenological Technique 9th ed Ann Arbor Edwards Brothers 1942
 Simmons J S and Gentzkow C J Laboratory Methods of the United States Army 5th ed Philadelphia, Lea & Febiger 1944
 Todd J S and Sanford A H Clinical Diagnosis by Laboratory Methods 10th ed Philadelphia W B Saunders Company 1943

SECTION XXIII

MEDICAL THERAPEUTICS

- Barr D P Modern Medical Therapy in General Practice 3 Vols Baltimore Williams and Wilkins 1940
 Beckman H. Treatment in General Practice 5th ed Philadelphia W B Saunders Company 1945
 Clausen E The Therapeutic Value of Radiation Medicine 8 419 (Dec) 1929
 Cook E F, et al Remington's Practice of Pharmacy 8th ed Philadelphia J B Lippincott Company 1936
 Epitome of the U.S.P. and N.F., Chicago American Medical Association 1944
 Goodman L., and Gilman A The Pharmacological Basis of Therapeutics New York, Macmillan Company 1941
 Hirschfeld S Wanger J J and Hyman H T Influence of Velocity on the Response to Intravenous Injections Arch of Int Med 47:250 (Feb) 1931
 Krusen F H. Physical Medicine The Employment of Physical Agents for Diagnosis and Therapy Philadelphia W B Saunders Company 1941
 New and Nonofficial Remedies Chicago American Medical Association 1945
 The Pharmacopeia and the Physician Chicago American Medical Association 1944
 Sollman T A Manual of Pharmacology 6th ed Philadelphia W B Saunders Company 1942
 Useful Drugs Chicago American Medical Association 1945

SECTION XXIV

SURGICAL THERAPEUTICS

- Babcock W W., et al Principles and Practice of Surgery Philadelphia, Lea & Febiger 1944
 Bunnell S Surgery of the Hand Philadelphia J B Lippincott Company 1944
 Burns Shock Wound Healing and Vascular Injuries National Research Council Committee on Surgery Philadelphia, W B Saunders Company 1943
 Callander C L Surgical Anatomy 2nd ed Philadelphia, W B Saunders Company 1939
 Christopher F Minor Surgery 5th ed Philadelphia W B Saunders Company 1944
 Christopher F (ed) Textbook of Surgery 4th ed Philadelphia, W B Saunders Company 1945
 Cole W H and Elman R Textbook of General Surgery 4th ed New York D Appleton Century Company 1944
 Cutler E C and Zollinger R. Atlas of Surgical Operations New York Macmillan Company 1943
 Davis L E The Principles of Neurological Surgery 2nd ed Philadelphia, Lea & Febiger 1942
 Ferguson L K Surgery of the Ambulatory Patient, Philadelphia J B Lippincott Company 1942
 Fundamentals of Anesthesia National Research Council Division of Medical Sciences 2nd ed American Medical Association 1944
 Hauser E D Diseases of the Foot Philadelphia W B Saunders Company 1939
 Jones T and Shepard W C Manual of Surgical Anatomy Philadelphia, W B Saunders Company 1945
 Kanavel A B Infections of the Hand 7th ed Philadelphia Lea & Febiger 1942
 Lewin, P The Foot and Ankle 2nd ed Philadelphia Lea & Febiger 1941
 Lundy J S. Clinical Anesthesia, Philadelphia, W B Saunders Company 1942
 Moorhead, J L. Traumatic Surgery Philadelphia, W B Saunders Company 1945
 Orr T G Operations of General Surgery Philadelphia W B Saunders Company 1944
 Thorek, M. Modern Surgical Technique 1 Vol Philadelphia, J B Lippincott Company 1943

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wasted time into useful purposeful activity that in December 1942 the Army Air Forces Convalescent Training Program was initiated in all Army Air Forces hospitals

Designed originally for the pre combat sick or injured soldier the program had a dual mission first, to send the soldier patient back to duty in the best possible physical condition in the shortest possible time second to teach the soldier patient something that would make him a more efficient and more effective fighting man With the inception of this program physical activity and military education became as much a doctor's prescription as drugs and diet Muscles were not permitted to atrophy, for reconditioning exercises started the moment the acute illness or surgery terminated Minds did not become stagnant for time was spent purposefully The classroom and gymnasium were moved into the wards and corrective exercise light reconditioning calisthenics training films radio code practice periods camouflage and discussions on why we fight and the postwar world became an integral part of the hospital day Radios carburetors exercise apparatus and even airplane engines were brought into the wards and sunrooms The hospital was transformed into a combination spa gymnasium schoolroom, machine shop and New England Town Hall

Early in the convalescent program it was noted that, to obtain maximum results reconditioning had to start *at the earliest possible moment* following acute disease or injury It had to be purposeful and progressive and graduated as the patient's physical tolerance increased Special corrective exercises had to be designed to meet specific military needs and experienced physical training instructors given special instruction in administering them A positive attempt was made to correlate ward fatigue and detail work with the soldier's disability to aid in functional recovery Men with hand finger and wrist injuries were given fatigue duties that involved finger and hand manipulation Those with bad backs or knees were given duties which involved bending stretching kneeling lifting and other movements which aided in strengthening the affected parts

The *Handbook of Recovery (AF Manual No 23)*¹ was prepared as a prescription blank for all types of orthopedic disabilities It included a nine page section on anatomy physiology and pathology written in simple language to show the soldier patient why he was in the program the value and effects of active exercise how bones muscles and nerves are made what happens when they are injured and how they heal This was followed by a series of two page prescription blanks for the specific injury demonstrating to the soldier patient by diagram the normal function of the injured part his diagnosis and disability, the type of physiotherapy prescribed the illustrated active exercises to be done with and without supervision and a personal objective recovery chart which the patient himself maintained This was an effective innovation as it not only gave the patient an insight into and understanding of his condition but great motivation through his ability to measure his own progress objectively An accompanying *Instructor's Manual for Physical Retraining (AF Manual No 24)* was prepared for the guidance and use of physical reconditioning instructors in order that they might properly administer and supervise the exercises to obtain maximum results

Let's Walk (AF Manual No 49)³ was another publication designed for the patient which met with widespread use and approval. Unique in its presentation this booklet dealt with the functional aspects of walking with the use of aids. It first gave the patient a brief psychological orientation to his disability and then proceeded to demonstrate objectively through the use of illustrations and charts the factors involved in walking with aids, the muscles which must be utilized and therefore strengthened to use walking aids effectively, the techniques and gaits of walking, practical hints on care of the aids, and most important, insight and motivation.

During the convalescent period the ambulatory patients were grouped together in special convalescent wards with a general practitioner as medical officer in charge. The role of the medical officer was more than just a supervisor over the daily sick call and routine rounds. He had the responsibility of classifying the patients in terms of their physical capacities, supervising both the physical reconditioning and educational activities, motivating the patients by giving them insight and understanding of the nature of their illness and disabilities, and practicing preventive mental hygiene through individual discussions of personal problems. His duties and activities were as diversified as those of the general practitioner in civilian practice, for he treated the patient as an individual and translated the patient's illness and disabilities in terms of their effects on his military, social, economic and personal problems. He was a family doctor in the truest sense of the word, for upon him rested the responsibility of bridging the gap between the bed and full military duty by a planned program of productive convalescence. The philosophy of the physical medicine program was recently reported by the author and Voldeng.⁴

More interesting, however, and perhaps more significant than the means used to meet the objectives of the convalescent program were the end themselves. Some of the pertinent observations of the Convalescent Training Program after over three years of experience with millions of man hours spent by thousands of patients were: hospitalization time was shortened, hospital readmissions were reduced, sick leaves were eliminated except in extraordinary cases, and the morale of the soldier patients was immeasurably improved when they were kept busy and interested in purposeful activity.

Clinical Studies and Their Results. 1. *Virus Pneumonia*.—A number of interesting clinical studies were made. Van Ravenswaay⁵ and his coworkers studied 645 cases of virus pneumonia which, after being treated in the same ward, were then assigned to alternate convalescent wards. In Ward I nature was permitted to take its course and men sat around until they and the medical officer felt they were ready for duty. In Ward II the patients were kept in bed until their sedimentation rate reached 10 mm in one half hour and then were put in a reconditioning program beginning with exercise for one half hour the first day and increasing progressively until the twelfth day when the patient was participating in a full six hour day of physical training, mass games, competitive sports and active recreation including a ten mile hike. Group I averaged 45 days hospitalization with a 30 per cent recurrence rate; the patients in Group II averaged only 31 days with but a 3 per cent recurrence rate; 45 days hospitalization, unsupervised, 31 days with graduated conditioning—a 30 per cent recur-

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Karpovich⁶ and his associates at the School of Aviation Medicine San Antonio, Texas studied a similar group of 200 aviation cadets convalescing from virus pneumonia. Using a modification of the Harvard Step Test and starting as early as the first afebrile day Karpovich found that by the reactions to this test it was possible to determine, with some degree of accuracy, the patient's ability to enter into and participate in an active convalescent program. An interesting by product of Karpovich's observations was that patients being tested required five days less hospitalization time than those participating in the general program thus making evident the tolerance and ability of these men to participate in an even more active program with beneficial results.

Marquardt⁷ at the AAF Regional Station Hospital No. 1 Coral Gables Florida made an interesting observation on a small group of patients recovering from virus pneumonia. These patients were clinically well but had residual persistently positive x ray findings. They were put on hyper ventilation every waking hour twelve times a day and serial x rays were made every twelve hours. Over ninety per cent of the patients in the series became x ray clear in a period of ninety six hours.

2 *Orthopedic Patients*—Early in the operation of the convalescent program it was noted clinically that orthopedic patients requiring fixation of a specific member did not if kept in top physical condition generally show the usual degree of muscular atrophy in the fixed part and that when the cast was removed could be reconditioned to duty in approximately one half the usual convalescent time. Whether this was due to increased blood velocity, exercise within the cast, vasomotor stimulation or lack of capillary loss gives food for thought and further investigation.

3 *Rheumatic Fever*—As a part of the AAF Rheumatic Fever Control Program Karpovich,⁸ Weiss, Starr and Ershler did a study on physical fitness testing and physical training of convalescent rheumatic fever patients. Because of the chronic recurrent nature of the disease and the possibility of disabling cardiac sequelae it was felt that special emphasis should be placed on the standardization of physical activity and convalescent training of these patients by working out a series of graduated physical fitness tests which could be used in conjunction with the usual clinical observation to determine the rate by which the patient could safely be permitted to progress to increased physical activity.

Using a modification of the *Harvard Step Test* with two benches 12 and 20 inches in height and a cadence of twenty four steps per minute physical testing was started when in the judgment of the medical officer the patient should be permitted to be out of bed in a chair. The first test involved twelve step ups in thirty seconds using the 12 inch stool. Criteria for passing that test was pulse rate less than 100 per minute one minute after the exercise and good coordination in performance. If the patient passed this test and the medical officer concurred he was allowed to be ambulatory in the ward. Using the same criteria the next test involved twelve step ups in thirty seconds using the 20 inch stool. In this test it was found that body metabolism in the average individual was raised eight to ten times. If this test was passed the patient

program of graduated work assignments. These exercises were given in sessions twice daily over a period of approximately nine weeks; they were then increased to thirty minutes twice daily. The exercises used at the beginning of the program were found to raise heart metabolism twenty-three times. The degree of improvement of an exercise during this period was gradually increased to a level which raised the metabolic rate seven to nine times the normal.

The next level of physical fitness testing was designed to progress from exercising of twenty-four step-ups per minute to a 20-minute stair for a period of five minutes. Stairing was based on an duration of an exercise

for the past five one minute after an exercise. If the patient made a normal passing score on this test and the medical officer in charge concurred, he was placed in the business portion of the program with a minimum stay of two weeks in each of the four periods. In the first period, physical training was given for five minutes daily; in the second period, five minutes daily; in the third, seven minutes daily; and in the final period, ten minutes daily. Intensity of exercise was increased in each period and progressions were made only with the concurrence of the medical officer in charge. Progressions from period one to period two were automatic after two weeks, but progressions from period two to period three were dependent upon the scores made on the progressive test. There were no occurrences of untoward reactions in any patient used in the study.

It is interesting to note that before a comprehensive rehabilitation program was instituted in the hospital approximately 85 per cent of patients with myocardial infarct were discharged from the service on medical grounds. After the inception of the program, accompanied by special recreation and educational training classes and selective placement, 85 per cent of the myocardial infarct patients were retained in the service and were returning to civilian life were credited as to their vocational positions and families.

4. Patients of All Types—The standard AAF Physical Fitness Test evaluates of chest-size, strength, and reaction against time was administered to five to eight patients of all types on their day of discharge from the hospital. These men made a "normal" score per cent lower scores than the average made by troops on duty status at the same bases, which conclusively indicated that men were leaving the hospital physically fit for duty.

5. The Duration of Bed Rest—During the war there has been tremendous interest in the governmental and private medical programs of bed rest and many authors have written on this subject. ¹⁰⁻¹² Kety,¹² working with observations of rest and starting them under carefully controlled conditions of complete bed rest for six weeks, in his preliminary conclusions has noted that these individuals reacted with a reduced approximately 2 per cent, that combination is markedly decreased, especially in the preconceptive senses, that blood volume is reduced from 10 to 25 per cent, and there is a reduction on the average of 11 per cent in the size of the heart. He noted that in the standard work test, after a period of bed rest, there is a reduction of cardiac effectiveness with a "resting" individual, both at rest and in work tests; work efficiency drops with a pulse rate of 125 on the first day after prolonged bed rest would

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Using a modification of the *Harvard Step Test* with two benches 12 and 20 inches in height and a cadence of twenty four steps per minute physical testing was started when in the judgment of the medical officer the patient should be permitted to be out of bed in a chair. The first test involved twelve step ups in thirty seconds using the 12 inch stool. Criteria for passing that test was pulse rate less than 100 per minute one minute after the exercise, and good coordination in performance. If the patient passed this test and the medical officer concurred he was allowed to be ambulatory in the ward. Using the same criteria the next test involved twelve step ups in thirty seconds using the 20 inch stool. In this test it was found that body metabolism in the average individual was raised eight to ten times. If this test was passed the patient was placed on a

In the convalescent hospital each patient was assigned to a designated physician who became his family doctor. A general practitioner in the broadest sense of the term, this personal physician was carefully chosen because of his training, experience, and inherent ability to understand physical and emotional disabilities. Many were given special clinical courses in the newer psychiatric diagnostic and therapeutic techniques. They were in reality specialists in the psychology of emotional stress and the sciences of organic repair. They were specialists in prescribing, evaluating, and supervising physical therapy and physical reconditioning which required the broadest clinical experience and skill. They had to have a basic knowledge of functional anatomy and physiology, psychiatry, psychology,

often cause the pulse to reach 170. He noted further that it takes a period up to sixty days to recover normal pulse rate for the same work load. A mounting pulse rate even at complete bed rest under basal conditions was also observed pointing to the increasing incapacity of the heart to maintain the circulation adequately. Keys also noted that it is very difficult to keep a patient in positive calcium balance especially during the early days of bed rest, and that more than twice the amount of protein in the diet is necessary to keep the patient in normal nitrogen balance during this period. There is also a marked increase in the urinary output of thiamine and riboflavin. Barr¹⁴ and his associates have found in general this same phenomena plus a marked hyperreaction to the tilt table.

6 Early Ambulation after Surgery—Powers¹ and Whipple¹⁰ have reported the response to early ambulation of two series of surgical patients who were up and about on the first or second postoperative day. Powers series was of 500 patients and Whipple's approximately 1000. The cases varied from simple appendectomies to subtotal gastrectomies, a large number of cases of biliary surgery were included in Whipple's series. The following is a broad summary of the results.

Patients who were permitted early ambulation averaged five days less hospitalization. In Powers series they got back to their usual jobs four weeks earlier than the parallel group who stayed in bed the conventional length of time. They required less than half the amount of sedation than the late ambulatory group. The postoperative complications were diminished about 50 per cent in the early ambulation group. They went on regular diet the second or third day. Lemas were cut about 80 per cent. No patient on early ambulation required catheterization after he became ambulatory. All of these observations are most significant and make us wonder whether some of our old conceptions and practices should not be re-evaluated.

Convalescence and Rehabilitation to Meet Individual Needs—When overseas combat patients started entering the hospitals in 1943 it was realized that the training program carried on for the pre-combat trainee as outlined did not meet their total needs and that special convalescence and rehabilitation following the completion of definitive care was necessary. The convalescent hospitals were designed to meet the needs of the whole man regardless of what those needs were—medical, physical, psychiatric, social, vocational, educational or personal. The physical reconditioning program emphasized competitive team play and active recreation including athletics, bicycling, horseback riding, fishing, swimming, hiking and skiing.

The educational program consisted of training in military specialties, academic courses, commercial education, general and specialized wood working, metal working and machine shop practice, welding, photography, electrical and radio repair and numerous other courses. Diversified activities such as journalism, radio broadcasting, dramatics, art, handicrafts and other avocational activities were more than diversional in that they provided the media along with personal counseling and a well rounded and complete recreation program of psychological readjustment and resocialization.

The purpose of the program was to provide the soldier patient with a laboratory of opportunity through physical reconditioning, educational, avocational pursuits and recreation whereby he might recover the attitudes, habits and values compatible with normal behavior patterns.

a place on the production line and prove that they could with good selective placement produce on a par with the normal workers. An example is the Ford Motor Company River Rouge plant where more than 11 000 handicapped civilian workers were employed.

During the war 83 per cent of our nation's industries employed handicapped workers. Those industries have reported that among the handicapped there has been a much smaller labor turnover, less absenteeism, fewer accidents and equal or higher production rates. Many employers before this vast experience with handicapped employees had a fear of increased accident rates. The industrial accident rate of 87 of the great industrial plants in America, each having from 50 to 12 000 handicapped employees, has disproved that theory. Their reports show that 56 per cent found the accident rate for the handicapped lower than that of the able-bodied, 42 per cent found the rate the same as for the able-bodied and only 2 per cent found it to be higher.

The answer to the utilization of handicapped workers lies not so much in fitting the job to the worker as in *fitting the worker to the job*. Through this procedure the worker in reality becomes non-handicapped as far as his particular job is concerned. It is not a matter of coddling but one of proper placement. The worker is fitted to the task and knows a full day's work is expected of him.

Most individuals use less than 10 per cent of their potential efficiencies in normal pursuits. It is only in emergencies that we call upon our tremendous reserves of physical power and ability. In many cases a worker's physical defect acts as a tremendous stimulus to overcompensation, resulting in extraordinary physical ability. Adler¹⁸ developed a complete system of psychology on the basis of inferiority. He believed that the successful, the efficient and the aggressive individual was one who was compensating for some inferiority. The employer of handicapped workmen is putting that psychology into purposeful and gainful application.

A survey completed in a number of Westinghouse Electric Company plants disclosed that approximately 19 per cent of the occupations could be performed satisfactorily by persons having only one leg, 17 per cent by operators who must use crutches, 83 per cent by men with only one eye and 82 per cent by the deaf. Although much work still needs to be done in analyzing the physical requirements of jobs and physical capacities of workers, the *United States Employment Service* has developed adequate tools and techniques to meet the needs of the bulk of our handicapped population.

Economic, Social and Moral Dividends from Rehabilitation.—The *National Office of Vocational Rehabilitation* has reported how rehabilitation pays off economically.¹⁹ Of the 43 997 persons undergoing vocational rehabilitation under this agency in 1944, 22 per cent (or more than 10 000) had never been gainfully employed and nearly 90 per cent were not employed at the time they started their rehabilitation. The average annual wage of the entire group prior to rehabilitation was \$148. After rehabilitation the average annual wage of the group increased to \$1 768. The total earnings of the entire group rose from \$6 510 556 to \$77 786 696.

Prior to rehabilitation the majority of these persons relied on general public assistance, not only for the disabled individual but also for his

ability, there was in addition to physical reconditioning educational training psychological adjustment and resocialization opportunities a complete program of vocational guidance based on achievement, aptitude and functional testing and interests determined by a series of short work experiences. In these short work experiences under a staff of experienced vocational instructors the patient had an opportunity to participate in a diversified educational program. He learned not only what he liked to do but what he was able to do both in terms of ability and in terms of any handicaps he had. He knew at the end of this period what he enjoyed doing what he was able to do and the occupations for which he possessed aptitudes. This information was then correlated by skilled vocational counselors with the job opportunities in the chosen field and the patient upon discharge from the hospital had a complete objective vocational profile and insight into his specific problem.

THE ART OF MEDICINE IN CIVILIAN REHABILITATION

The rehabilitation programs of the armed forces have demonstrated what can be done for the physically and emotionally wounded and handicapped to make them self sufficient self respecting employable persons. This has further been enhanced by the remarkable record of the handicapped and disabled in industry during the war.

Both the medical profession and the general public are genuinely concerned with the problems which must be faced by thousands of our disabled veterans as they return to their communities. These men have earned the best that a grateful nation can give them. Few of us realize however that our disabled veterans constitute only a small part of the total number of handicapped and disabled in this country.

We had in 1940 in the United States some six and one half million disabled males between the ages of 15 and 64 years men who normally would be income producing. These six and a half million plus the number of disabled or handicapped veterans being discharged from our military hospitals will give us when demobilization is completed approximately eight million working age males who are disabled to the extent of requiring physical or vocational rehabilitation or special placement aids if they are to be successfully employed. This represents one person in sixteen in our general population and one in seven in our male working population. The number of persons who are permanently disabled by accident alone increases by 350 000 each year. The total problem has been summarized by Donahue and Tibbitts¹⁷

During the first four years of war there were approximately 17 000 amputations in the Army but during this same period there were 120 000 major amputations from disease and accidents in our civilian population.

The armed forces and some outstanding community and civilian agencies such as New York's Institute for the Crippled and Disabled have demonstrated that the physical and emotional rehabilitation of our handicapped and disabled is possible. Some authorities have estimated that up to 97 per cent of all our handicapped can be rehabilitated to such an extent that they can be gainfully employed.

Handicapped Workers in Industry—The short labor market during the war gave many disabled persons the first opportunity of their lives to take

One such group is the *Baruch Committee on Physical Medicine* which is translating the experience of the armed forces in rehabilitation into its civilian application in order that this experience may be made available to the medical profession and those interested in community rehabilitation centers. They are blueprinting a model center outlining its mission, organization, components, physical set up, and its relationship to the medical and allied professions—industry, labor, social, and governmental agencies—and to the community as a whole.

Adequate programs designed to take men from the bed back to the job will not only save the worker as a producing member of the community but can save industry tremendous sums from compensation claims. The expenditure of a few dollars on retraining and rehabilitation, as has been shown, pays high dividends. In plants in smaller communities, part-time rehabilitation assistants can make valuable contributions. The athletic director in the school, vocational teachers, and other trained members of the community can be organized to do a functional team job on a part-time basis, working under the direction of an industrial surgeon to give a limited but most valuable rehabilitation program.

Opportunities for Workers in the Field of Rehabilitation—Many returning veterans have had invaluable in-service experience in this field and are eager to continue in the field of services to the handicapped. The *National Society for Crippled Children and Adults* has set up a personnel roster of persons trained in all fields of rehabilitation and is acting as a clearing house between the individual and the community to aid in meeting the needs of both.²⁰

The General Practitioner's Opportunity.—One of the greatest problems in rehabilitation is that of motivation—encouraging and convincing the disabled that they can rehabilitate themselves. This motivation to be the most effective must begin at the earliest possible moment following the accident or illness. The general practitioner who knows something of rehabilitation can start at the time of accident or crippling disease to allay the fears of the patient by giving him understanding, courage, and hope predicated on an accurate knowledge of what can be done.

The general practitioner, if he is to meet the needs of his patients, must know something about rehabilitation and selective placement of the handicapped. He must interpret to his patients the findings of the specialists in words that are understandable and meaningful. The burden of explaining to the patient the nature and extent of his disability falls upon the family doctor. That explanation cannot be in medical terms of the disability alone but must be in terms of its effects upon the vocational, social, economic, family, and personal life of the patient.

For example, let's take the hypothetical case of Bill Jones, who is fifty years old, a brakeman on a railroad. He enters the hospital with his first cardiac breakdown. He is examined by the resident staff, the diagnosis is made, and checked by the visiting physician, and proper treatment instituted. After a short period, his heart becomes recompensated, and he is medically discharged with usually this advice: "Take your medicine regularly, come back to the clinic every two weeks, and you know of course that you won't be able to do any strenuous work." The bottom drops out of Jones's world. He says to himself, "I can't stop. I have two kids in high school, my house isn't paid for, my insurance won't be paid

family The annual cost of this assistance to the taxpayer was from \$300 to \$500 per case but the total cost of their rehabilitation averaged only \$300 per case a non recurring expenditure

No degree of economic gain however can measure the social and moral satisfactions obtained by the successfully rehabilitated and employed handicapped worker and his family Nor can it measure the value to society in the transformation of these individuals from dependents to productive self reliant individuals

The handicapped citizens proved their worth during the war and were lauded by industry for their efforts As production is cut back, however industry will drop large numbers from its payrolls Demobilized veterans are returning from the war in increasingly large numbers and more employees have to be released to make room for veterans who not only deserve jobs but are guaranteed them by law Dismissals are on the basis of seniority and the disabled being the last to be hired are among the first to be dismissed

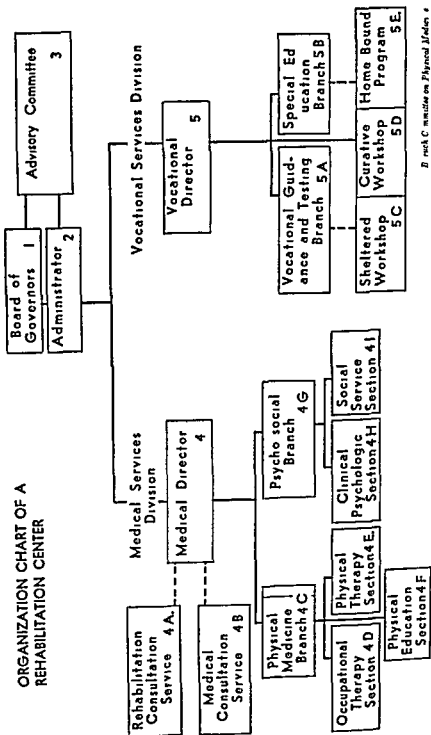
The handicapped worker after enjoying a period of economic prosperity suddenly finds that the labor market is loosening and that jobs for which he can qualify are no longer available Greater production during the war allowed industry to break job structures into small components but now as production is being curtailed the average worker instead of doing one small highly specialized task finds he must be able to do several This demand for greater flexibility on the part of the worker further reduces the employment opportunities of the disabled During the war the job was adapted to the worker now the worker must be made to fit the job The only answer lies in rehabilitating the worker

Vocational Rehabilitation Programs—The record of the federal state rehabilitation programs under the Office of Vocational Rehabilitation has been heartening but when it is compared to the one and one half to two million persons who are in urgent need of such care it is woefully inadequate The question usually raised is If the vocational rehabilitation program has been so successful with this small group why aren't the remaining million and a half to two million given its advantages?

The answer is that there simply aren't enough rehabilitation centers in the country to which these cases can be referred The state and federal rehabilitation programs do not do the actual physical restoration and vocational retraining of these people They supply the funds help select a suitable vocation for which the person possesses both aptitude and physical abilities and supervise his retraining The actual work however is done by other agencies These agencies may be public or private schools trade schools, apprenticeship training courses rehabilitation centers hospitals group clinics or other medical facilities Those who possess the necessary skilled personnel and facilities to do an adequate job of physical restoration and retaining and to rehabilitate cases vocationally are so few in number that opportunity is denied to the vast majority of our disabled population

The funds and authority are available but the facilities equipped for doing the actual job of rehabilitation are inadequate Several national organizations are making efforts to stimulate public interest in rehabilitation by demonstrating to both the medical profession and the public, what actually can be done by an integrated program of physical mental and social vocational adjustment

- 1 The Board of Governors should be composed of capable interested in financial leaders in the community who will set the policy and operating procedures of the Rehabilitation Center
- 2 The Administrator (medical or lay) plans, directs and supervises the activities of the Rehabilitation Center
- 3 The Advisory Committee should consist of representative committees of the (a) medical profession (b) industry (c) labor (d) social agencies (e) civic organizations. These committees will act as special advisory groups to the Board of Governors and will advise on specific problems in their respective fields
- 4 The Medical Director plans, directs and supervises the medical activities of the Medical Service Division maintains liaison with appropriate medical and social agencies operates and supervises the Rehabilitation Consultation Service plans, supervises and directs clinical investigation and research projects
- 4a The Rehabilitation Consultation Service provides consultation service to hospitals and physicians for the purpose of evaluation and prognosis of the patient's disability and the institution of a nearly rehabilitation program in the definitive stage of treatment, in cooperation with the physician and hospital Physical Medicine Department
- 4b The Medical Consultation Service should be composed of consulting medical specialists to provide special medical consultation for patients in the Rehabilitation Center
- 4c The Physical Medicine Branch should be headed by a Chief (a physician who is a specialist in the field of physical medicine and rehabilitation) who plans, directs and supervises all projects in the field of physical medicine plans and directs special investigation in the field of physical medicine maintains liaison with the Psycho-Social Vocational and Educational Services Branches maintains liaison with appropriate civilian and medical organizations relative to the professional problems pertinent to rehabilitation and physical medicine
- 4d The Occupational Therapy Section should be headed by a Chief (a qualified occupational therapy technician) who plans, directs and supervises the occupational therapy program for the Physical Medicine Branch and maintains liaison with the Chiefs of the Physical Therapy Section should be headed by a Chief (a qualified physical therapy technician) who plans, directs and supervises the program of physical therapy and maintains close liaison with the Chiefs of OT and PE Sections
- 4f The Physical Education Section should be headed by a Chief (a qualified specialist in physical education corrects and supervises an individual retraining) who plans, directs and supervises the program of physical education corrects and supervises the activities of the Physical Education Branch and maintains liaison with the Chiefs of the Physical Therapy Section should be headed by a Chief (a qualified physical therapy technician) who plans, directs and supervises the program of physical therapy and maintains close liaison with the Chiefs of OT and PE Sections
- 5 The Vocational Director plans, directs, and supervises the activities of the vocational and educational service division conducts vocational and psychological testing research activities operates and directs the vocational and educational activities of Sheltered Workshops, Curative Workshops and the Home-Bound program
- 5a The Vocational Guidance and Testing Branch should be headed by a Chief (a qualified specialist in vocational guidance) who plans, supervises and directs the activities of this branch operates a program of psychological testing vocational education and retraining conducts special investigative procedures in the field of vocational guidance and testing as it relates to physical and mental disabilities maintains close liaison with the Physical Medicine and Psycho-Social Branches
- 5b The Special Education Branch should be headed by a Chief (a qualified educator) who plans, directs, and supervises the activities of this branch including special education projects for the physically and mentally handicapped and maintains close liaison with the Vocational Guidance and Testing Branch
- 5c The Sheltered Workshop should be a workshop for the severely physically and mentally handicapped who are not capable of employment through usual industrial channels
- 5d The Curative Workshop should be a workshop the purpose of which is to coordinate physical and psychological disabilities with vocational opportunities and provide retraining in this specialized field
- 5e The Home-Bound program should provide for the individuals who are not ambulatory and must carry on in industrial projects within the confines of their own homes



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